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Report on the Epidemiology of Rheumatic Infection in South Australia.

FOREWORD.

THIS report describes the research into the subject conducted under the auspices of the Central Board of Health of South Australia and assisted by a grant for the purpose from the National Health and Medical Research Council (Australia).

The committee directing the work was: Dr. A. R. Southwood (Chairman), Dr. W. Christie, Professor J. Burton Cleland, Dr. R. Thorold Grant, Professor E. Weston Hurst, Dr. L. W. Jeffries, Dr. E. Britten Jones, Dr. J. W. Rollison. The Central Board of Health greatly appreciates the work conducted in an honorary capacity by the members of this committee.

The research officer was Dr. C. B. Sangster, by whom the report has been prepared.

The report was adopted by the committee at its meeting on October 6, 1939, and by the Central Board of Health on October 12, 1939.

(Sgd.) A. R. SOUTHWOOD,
Chairman, Central Board of Health.

INTRODUCTION.

In 1934 Dr. E. Britten Jones delivered his presidential address before members of the South Australian Branch of the British Medical Association, his subject being "The Rheumatic Child". He urged an investigation into the occurrence of juvenile rheumatism in South Australia, and suggested that the varying climatic conditions in parts of the State, the frequency of rheumatic infection in certain areas and the apparent severity of cases coming from sparsely populated country districts all offered a field for research.

At the Science Congress (the Australian and New Zealand Association for the Advancement of Science) in Auckland in January, 1937, Dr. A. R. Southwood spoke on "Heart Disease and National Welfare" in his address as President of the Section of Medical Science and National Health. The association subsequently adopted a resolution:

That this association, recognizing the influence of diseases of the heart on national welfare, recommend to the Governments of the Commonwealth of Australia and

the Dominion of New Zealand: (*a*) that research into this subject be encouraged and promoted by the National Health and Medical Research Council and similar bodies, (*b*) . . .

The first session of the National Health and Medical Research Council was held at Hobart in February, 1937. At this a resolution was passed, in reference to rheumatic infection and heart disease, urging the Commonwealth and State health departments to investigate the incidence of rheumatic infection and the possible causative factors.

In March, 1937, a committee was appointed by the Central Board of Health to make preliminary inquiries into the prevalence of rheumatic infection in South Australia. This committee submitted a report to the second session of the National Health and Medical Research Council, held at Canberra in June, 1937. In the report it was recommended that a part-time research officer be appointed to work under the direction of the committee appointed by the Central Board of Health, the person appointed to:

1. Conduct a rheumatic survey.
2. Investigate home conditions of sufferers from rheumatic infection.
3. Investigate especially the incidence of rheumatic infection in northern areas of the State.
4. Investigate the incidence of rheumatic infection among aborigines in the State.
5. In cooperation with the Director of the Institute of Medical and Veterinary Science, investigate rheumatic disease from the serological point of view in town children and country children.
6. Endeavour to discover why country children are especially prone to the severe types of infection.

At its third session in November, 1937, the National Health and Medical Research Council made a grant available to the Central Board of Health of South Australia to assist the Rheumatism Committee in the investigation.

The main portion of the research has been conducted during 1938 and the early months of 1939. The findings are presented in this report under the following sections:

1. The analysis of the Adelaide Hospital and Adelaide Children's Hospital records.
2. The distribution of juvenile rheumatism in the city of Adelaide and suburbs.
3. Juvenile rheumatism in country areas of the State.
4. Home conditions of patients with juvenile rheumatism.
5. Mortality statistics.
6. The seasonal occurrence of acute rheumatism and the influence of rainfall and temperature on the prevalence of the disease.
7. Experimental and laboratory investigations.
8. Rheumatic infection in aborigines.

Throughout this report the term "juvenile rheumatism" includes the conditions of acute and subacute rheumatic fever, rheumatic carditis and Sydenham's chorea, whether occurring in children or adults. The term is a convenient one to employ to embrace all cases, for the disease, although differing in the frequency of certain manifestations, is essentially the same whatever the age of the patient.

In the course of the investigation the consideration of some clinical and ætiological factors has appeared of sufficient importance to warrant detailed consideration. The appendices to the report embody the following material: (i) rheumatic nodules and their significance, (ii) skin rashes in acute rheumatism, (iii) the distribution of movements in chorea, (iv) the colouring of children in juvenile rheumatism, (v) the family history in juvenile rheumatism, (vi) trauma as a factor in the ætiology of juvenile rheumatism.

The investigation was fortunate in its friends. It is not possible to thank all who helped, but some whose aid was particularly valuable must be mentioned. The maps and graphs in the report were constructed by the State Lands and Survey Department, under the supervision of Mr. S. Symons. The Government Statist, Mr. A. W. Bowden, and his staff kindly supplied figures relating to death returns, and Mr. E. A. Cornish, of the Waite Agricultural Research Institute, gave valuable assistance in evolving certain correlation coefficients. The former Director of Education, Mr. W. J. Adey, and the Principal Medical Officer of the Education Department, Dr. W. Christie, cooperated in the arrangements for the examination of country school children. The almoners attached to the Adelaide Hospital and Adelaide Children's Hospital, Miss M. T. Noal and Miss J. Lupton respectively, investigated the home conditions of rheumatic patients, and the records staff of each hospital spent much time in seeking out case records for analysis. The Director of the Institute of Medical and Veterinary Science, Professor E. Weston Hurst, with Professor A. E. Platt and other members of his staff, are to be thanked for their work in connexion with the laboratory and experimental investigations. The Director-General of Medical Services, Dr. L. W. Jeffries, kindly arranged for *questionnaires* to be sent to country hospitals throughout the State. Thanks are also due to the members of the honorary staffs of both hospitals for allowing access to patients and case notes, and to members of the South Australian Branch of the British Medical Association for their cooperation. The ready response of all whose help was sought enabled the investigation to proceed in a smooth and orderly manner, and for this the Rheumatism Committee is very grateful.

The Analysis of Hospital Records.

One of the main sources of material for this investigation has been the records of patients suffering from acute rheumatism and chorea who were admitted to the Adelaide Hospital and Adelaide Children's Hospital over the eleven-year period 1928-1938. Practically all patients admitted because

of these conditions during 1938, and also those until June, 1939, were personally observed and the clinical course of the disease was noted. A critical analysis of the records was made, and only cases of active rheumatic infection have been included in the study. The figures given do not refer to entirely separate cases, although most of them do. A number of patients were readmitted several times to hospital because of active rheumatism, and, where it has been thought necessary to subdivide or classify these cases, an explanation to this effect has been made. The patients were arbitrarily divided into three groups: (i) those who suffered from acute rheumatism alone; (ii) those who suffered from chorea alone; (iii) those who, either on admission or at some time during the period in hospital, showed manifestations of both acute rheumatism and chorea.

Admissions.

The Adelaide Children's Hospital.

There were 450 admissions to the Adelaide Children's Hospital on account of juvenile rheumatism during the eleven years, the number varying between 31 in 1932 and 52 in 1935. The average number of admissions per year was approximately 41. The number of patients admitted in a first attack was 305. These 450 cases comprised:

Acute rheumatism	290 or 64·5%
Chorea	132 or 29·3%
Acute rheumatism combined with chorea	28 or 6·2%

Over the ten-year period 1928-1937 the total number of admissions from all causes was 22,983, and the total number of patients admitted because of acute rheumatism or chorea was 409. Thus 1·8% of the admissions to the hospital were on account of an active rheumatic infection. This figure may be contrasted with that calculated from figures given by Boyd Graham.[1] An estimate, based on his figures, disclosed that 2·5% of all patients admitted to the Children's Hospital in Melbourne were suffering from acute rheumatism or chorea. The figures for the two hospitals are comparable, for both were obtained after perusal of the individual case records. It appears from these figures that juvenile rheumatism is less prevalent amongst children in Adelaide than it is in Melbourne.

The Adelaide Hospital.

There were 390 admissions to the Adelaide Hospital during the same period on account of juvenile rheumatism, the number varying between 19 in 1932 and 65 in 1936; the average number of admissions per year was approximately 35. The number of patients admitted in a first attack was 218. These 390 cases included the following:

Acute rheumatism	327 or 83·8%
Chorea	58 or 14·9%
Acute rheumatism combined with chorea	5 or 1·3%

Working out similar figures to those given above for the Children's Hospital, the percentage of cases of active rheumatic infection to total admissions was as follows:

Adelaide Hospital	0·33%
Royal Melbourne Hospital	0·29%
Alfred Hospital	0·26%

Homer Swift[2] mentions that "rheumatic fever in the form of acute rheumatic polyarthritis makes up from 3 to 7% of all cases admitted to large general hospitals in the United States and Europe". It appears that the disease is less prevalent in Adelaide and Melbourne than in those cities of the United States and Europe from which Swift derived his figures.

There was a drop in the percentage admissions on account of chorea to the Adelaide Hospital as compared with the Children's Hospital; this manifestation of juvenile rheumatism was approximately twice as common in children under twelve as compared with persons over this age.

Sex Ratio.

There was a negligible difference in the sex ratio of the 450 patients admitted to the Children's Hospital, 224 of them being males and 226 females. The corresponding Adelaide Hospital figures showed that only a slightly greater proportion of females were affected by the disease than males, for of the 390 patients admitted, 211, or 54%, were females and 179, or 46%, were males. It was found that acute rheumatism occurred almost equally in the sexes, and chorea slightly more commonly in females.

Investigators have found that mitral stenosis as a complication of juvenile rheumatism is more common amongst females than males. Britten Jones,[3] from an analysis of the Adelaide Children's Hospital records for the years 1928-1933 inclusive, found that, although the numbers of boys to girls leaving the hospital with a bruit after an attack of juvenile rheumatism were almost equal, when those with definite evidence of mitral stenosis were picked out there were more than twice as many girls as boys.

Age Groups.

It is well recognized that juvenile rheumatism reaches its maximum incidence during the school-going ages; but its occurrence before the age of five is not uncommon. With the object of finding out at which ages frank manifestations of juvenile rheumatism most often made their initial appearance, only those patients admitted in a first attack were considered. There were 523 such patients, and the numbers in the different age groups are shown in Chart I.

In this series the ages of highest incidence of first attacks were between five and twelve, although right up to the age of sixteen the incidence of initial attacks was high. Thereafter the numbers rapidly

fell, and with increasing age the incidence became less. The commonest age for the first attack was between seven and ten years.

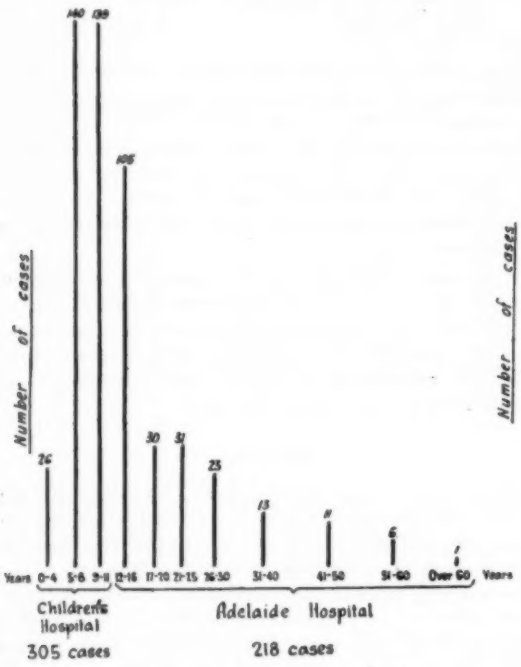


Chart I.

Showing admission of patients suffering from acute rheumatism and chorea to the Adelaide Children's Hospital and the Adelaide Hospital, 1928-1938, in age groups (first attacks).

Mortality.

At the Adelaide Children's Hospital there were 19 deaths among 405 patients admitted with active juvenile rheumatism. These figures refer to those admitted in a first attack, or those in whom a previous uncomplicated attack had occurred. The immediate mortality rate was thus 4·69%. Acute rheumatism accounted for 17 of the deaths, and acute rheumatism combined with chorea for two; it is of interest to note that no deaths occurred from chorea alone.

At the Adelaide Hospital, among the corresponding 309 patients, which include those above the age of twelve, there was only one death, and that from acute rheumatism.

For the total of 714 patients of all ages there was a mortality rate of 2·8% in the acute phase of the infection. The significant drop in the mortality rate between the two hospitals serves to indicate the very much greater severity of the infection in young individuals, and stresses how, in children, the infection produces its major effects upon the heart and not upon the joints and fibrous tissue structures. The remark of the French physician Lasègue that "rheumatism is a disease which licks the joints but bites the heart" is especially applicable to children.

The above findings referring to the mortality rate in the two hospitals may be compared with those given by other observers. Atwater[4] mentions that rheumatic fever kills few in its acute form. The case fatality rate, the number dying in an acute attack per hundred cases he worked out at 1·7. This figure was obtained from an estimate of the death rate in over 20,000 cases from different sources, the case fatality rate in the separate series ranging between 0·4% and 3·7%. Glover,[5] in his Milroy Lectures in 1930, states that:

> The immediate mortality of acute rheumatism is very low, generally under 1%. Even in hospitals where the worst cases are admitted the case mortality seldom exceeds 2%, save in children's hospitals, where the case mortality has reached 13% in certain outbreaks.

Causes of Death.

Among the 20 fatal cases mentioned, the commonest cause of death was heart failure, which followed fairly rapidly on a pancarditis. This occurred in nine instances. The immediate cause of death of four patients with a severe degree of carditis was pneumonia, of the lobar type in three instances and bronchopneumonic in one. In three instances death was due to congestive cardiac failure coming on at some considerable time after the onset of pancarditis. The causes of death of the remaining four patients were as follow: (*a*) cerebral hæmorrhage occurring in the acute phase of the infection, (*b*) septicæmia following on an attack of acute streptococcal tonsillitis whilst the patient was in hospital, (*c*) pulmonary embolism in a patient with mitral stenosis, (*d*) jaundice and cholæmia progressing to coma in a patient with pancarditis.

Cardiac Damage in Juvenile Rheumatism.

The cardiac damage sustained by patients admitted to the two hospitals was noted. This was assessed on the clinical findings recorded in the notes. Particular attention was paid to whether or not a murmur signifying an established cardiac lesion was present at the time of discharge of the patient from hospital. In this estimation the patients were followed up through the records of the out-patient department or through those of any subsequent admissions to hospital. The figures given deal only with patients admitted in a first attack.

Of 523 patients, 179, or 34%, were judged to be discharged with cardiac damage, a figure which stresses the importance of regarding the disease as being essentially one of the heart. The accepted fact that cardiac complications are much more frequent in the younger ages was borne out. It was found that no less than 40% of the 305 patients admitted to the Children's Hospital were discharged with a murmur significant of valvular disease, whereas the corresponding figure for patients over the age of twelve admitted to the Adelaide Hospital was 25%.

Cardiac damage was found to have followed acute rheumatism much more often than it did chorea. Among the 523 patients, 36% of those affected with acute rheumatism were judged to have some valvular disease at the time of their discharge from hospital, whereas the corresponding figure for cases

of chorea was 22%. However, the worst "cardiac cripple" met with among the patients seen personally over a period of sixteen months was a boy, aged thirteen years, who had combined mitral and aortic valvular disease and adherent pericardium of a severe degree, all the result of one attack of chorea at the age of seven.

Cardiac damage was found to be especially common in those patients manifesting both acute rheumatism and chorea, 13 out of the 22 being judged to have sustained valvular disease. Apparently the occurrence of the two manifestations of the disease in the one patient represents a more virulent or generalized infection, so that there is a greater liability of cardiac damage.

Among the 523 patients it was judged that mitral valvulitis occurred in 29·4%, mitral and aortic valvulitis in 1·5%, and not one sustained a pure aortic lesion. The great rarity of rheumatic aortic valvular disease by itself as a *post mortem* finding is known. Pericarditis occurred in only 3·3% of the patients. Of the 17 who suffered from this complication, 15 were under the age of twelve, which fact again stresses the greater severity of the infection in children.

Upper Respiratory Infections and Pharyngeal Sepsis.

It is well known that an upper respiratory infection, especially acute hæmolytic streptococcal tonsillitis, often precedes an attack of acute rheumatism. This has been stressed by Coburn[6] in America, and, as Schlesinger[7] has pointed out, there is commonly a "silent interval" of ten to twenty-one days between the tonsillitis and the rheumatic attack. Acute rheumatism, and particularly a relapse of this disease, may be preceded by other upper respiratory infections, like coryza, nasopharyngitis, and mild influenza, when these are associated with hæmolytic streptococci.

The relationship between hæmolytic streptococcal infection of the upper air passages and acute rheumatism has been investigated by Sheldon,[8] Collis,[9] Coburn and Pauli,[10] Bradley,[11] Thompson,[12] Gibson, Thompson and Stewart,[13] and many others.

In the case records studied attention was paid to a history of some upper respiratory infection preceding the onset of the attack. These infections are often mild and are liable to be overlooked in the history unless special inquiry is made for them. Consequently the figures quoted are a conservative estimate of the number of patients admitted to hospital in whom an upper respiratory infection, such as mentioned above, preceded the rheumatic attack.

In 34·2% of patients admitted to the two hospitals on account of acute rheumatism there was some preceding upper respiratory infection, generally acute tonsillitis. The corresponding figure for patients admitted with chorea was only 10·5%. These figures stress the importance of throat infections in the ætiology of the disease, and especially in acute rheumatism.

The portal of entry of the infecting agent in juvenile rheumatism is regarded as being the mucous membrane of the upper air passages, and especially the lymphoid tissue of the pharynx. Of 450 patients admitted to the Children's Hospital suffering from juvenile rheumatism, 46·7% were judged to have infected tonsils; the corresponding figure for the 390 admissions to the Adelaide Hospital was 22·1%. The difference in the percentages is largely explained by the practice of enucleation of infected tonsils during the younger years. Tonsillectomy in the "pre-rheumatic stage" by no means affords complete protection against the disease, but it lessens the liability to it, by minimizing severe recurrent throat infections.

Rheumatic Nodules.

Rheumatic nodules are indicative of severity of infection, and for this reason their occurrence was noted in the 840 patients admitted to the two hospitals. Of the 450 Children's Hospital patients, 20 (or 4·4%) had nodules; of the 390 Adelaide Hospital patients, in only 2 (or 0·6%) were nodules seen. The distribution of the 20 Children's Hospital cases was as shown in Table I.

Table I.

Condition.	Number Admitted.	Patients with Nodules.
Acute rheumatism	290	17 or 5·8%
Chorea	132	1 or 0·7%
Acute rheumatism combined with chorea	28	2

The difference in the severity of rheumatic infection in individuals above and below the age of twelve, and in acute rheumatism and chorea, is shown by the relative frequency of the occurrence of nodules. There was a high case fatality among the Children's Hospital patients, four of them dying. It is significant also that every patient who exhibited nodules was judged to have been discharged from hospital with cardiac damage. Further mention is made of rheumatic nodules in Appendix I.

Juvenile Rheumatism in Adelaide and Suburbs.

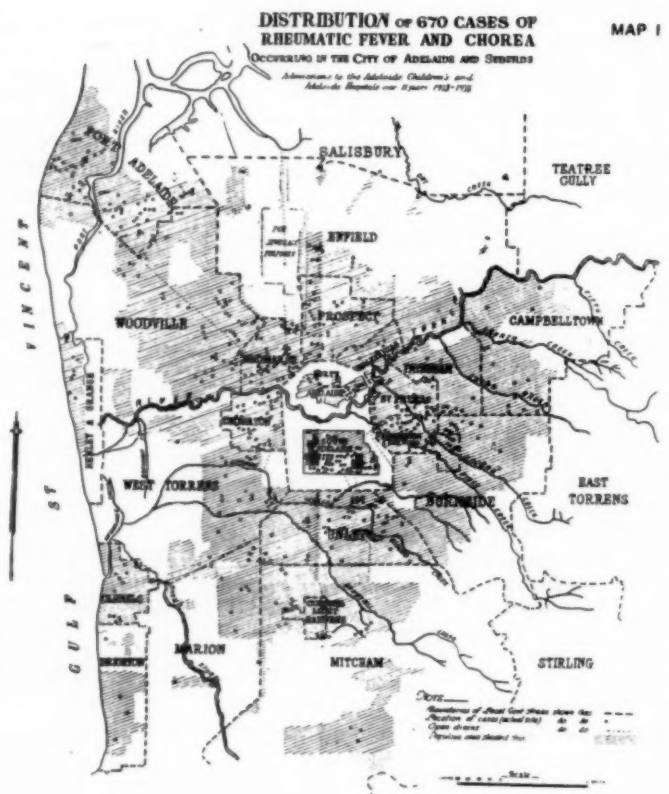
A "spot map" of the metropolitan area, showing the distribution of separate cases of acute rheumatism and chorea in patients admitted to the Children's and Adelaide Hospitals over the eleven-year period 1928-1938 was constructed. The number of cases dealt with was 670. In each case the actual site of the home was plotted, and 523 represented initial attacks. In most of the remainder it was possible to plot the site of the first attack. Map I is the result. The map does not represent the distribution of all cases occurring in the metropolitan area, but only those patients of the public hospital class, who were ill enough to merit admission for institutional treatment. It was sought to learn whether any particular area or place was more heavily "infected" than others; and, if so,

whether such factors as the proximity to creeks or rivers, the density of population, or the incidence of poverty played an ætiological role.

Considering first the areas shown on Map I to be more heavily "infected" with the disease, several of these are prominent. Outstanding is South Adelaide from which the greatest number of cases was derived. Other areas in which a large number occurred are Kensington and Norwood, Hindmarsh, Thebarton, Port Adelaide, parts of Prospect and Unley, St. Peters, and the part of Woodville in proximity to Hindmarsh.

The Influence of Proximity to Watercourses.

In the map all the creeks and rivers traversing the metropolitan area have been marked, and similarly the larger open drains. The creeks derive their water from the Mount Lofty ranges in proximity to Adelaide, and are shown as starting from the foothills. Five of them empty themselves into the River Torrens, and two of them traverse the southern portion of Adelaide, unite and empty their water into the Patawolonga Creek, together with the Sturt River. The Port River, which runs through the district of Port Adelaide, is a tidal river. All the creeks carry an appreciable volume of water in the winter months, and flow periodically at other times.



Certain more heavily "infected" areas lie in proximity to these watercourses, such as the districts of Kensington and Norwood and St. Peters, through which run the First and Second Creeks. A considerable proportion of the community in these districts belongs to the public hospital class. Through the Unley district, in which a fair proportion of cases occurred, runs the Glen Osmond Creek, joining with another to form the Keswick Creek. It may be said that, although quite a large portion of Unley contains people of the poorer class, those patients admitted to one or other of the hospitals on account of juvenile rheumatism were derived from homes reasonably close to a creek. A definite group of cases is shown in the West Torrens district, near the border of the Unley district. At this part the Brownhill and Keswick Creeks approach each other, the latter running through the "infected" area. In the district of Campbelltown a group of cases is seen to have occurred near the Third Creek, and groups occurred in those portions of Hindmarsh or Thebarton which are near the River Torrens.

The interpretation of the map is difficult. The rheumatic zones shown have not only to be considered in reference to watercourses, but also to the density and class of population. In several districts through which no river or creek runs, such as Prospect, the greater part of Hindmarsh and Thebarton, Woodville and most of Port Adelaide, the incidence of juvenile rheumatism is relatively high, and other factors doubtless operate.

At Prospect it was found on inspection of the places in which the majority of cases had occurred, that they were situated in a relatively low-lying damp part, towards which a lot of water gravitates after rain. A considerable portion of Hindmarsh and Port Adelaide might also be described as low-lying and damp, for water tends to lie about there in the winter months.

To sum up, it does not appear that creeks or rivers have a noticeable influence on the prevalence of the disease in Adelaide and suburbs. The impression gained from personal visits to the areas in which the rheumatic incidence was shown to be high, was that, in some at least, dampness of the ground from the lying about of water in the winter months might be incriminated as having favoured the occurrence of the disease.

The results of this investigation in Adelaide do not correspond with those of Thompson,[14] who, after making a spot map showing the distribution of rheumatic infection of children in Birmingham, came to the conclusion that the situation of working-class houses close to watercourses was of considerable importance in the production of a high incidence of juvenile rheumatism.

The Effects of Density of Population and of Poverty.

Map II and Table II (from which it was constructed) illustrate these aspects.

The estimated population and the proportion of it on relief, which was taken as a guide to the poverty existing in a district, have been calculated from the census figures of 1933, supplied by the Government statist, and the figures of the numbers on relief in 1933, supplied by the Unemployment Relief Council. The separate figures relating to the areas were available. The City of Adelaide has been considered by itself and as subdivisions, as shown. It was decided to divide South Adelaide arbitrarily into parts north and south of Grote and Wakefield Streets, for by far the greater part of the residential area of South Adelaide is south of these streets. The figures in the final column give the proportion of total cases of juvenile rheumatism in the estimated population of an area.

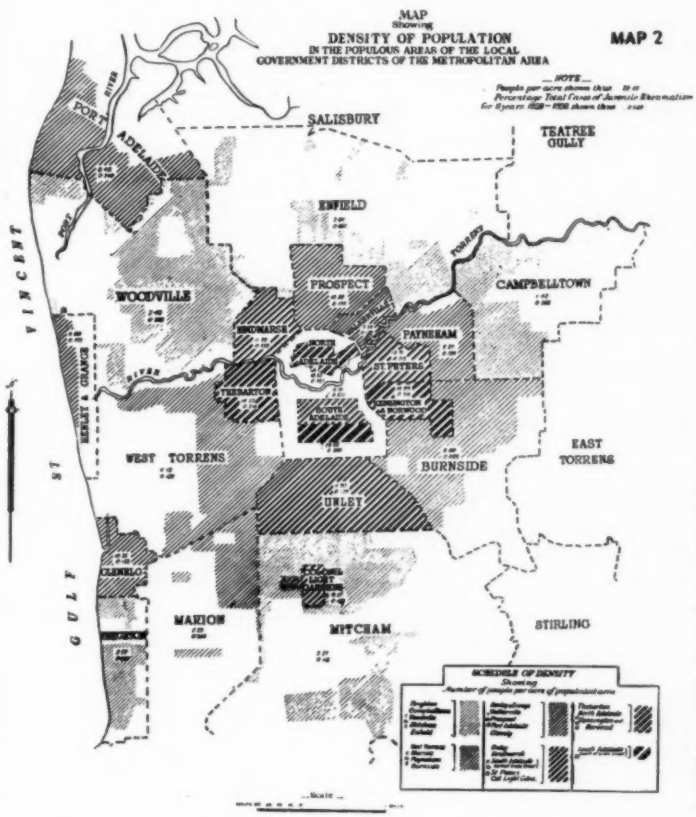
In some areas the density of population and the percentage total cases are both high, as in that portion of South Adelaide south of Grote and Wakefield Streets and the city of Adelaide as a whole. Similarly, in other areas, such as Mitcham and Brighton, the figures are low in reference to both factors. Enfield and Campbelltown, the lowest two areas on the list of density of population, both show a high percentage total cases, Enfield being third on the list. Other areas in which the relationship appears to break down, but not to the same extent as at Enfield and Campbelltown, are Unley, Colonel Light Gardens, Woodville, Burnside and West Torrens.

The impression formed on these results was that if a general relationship between the density of population and the percentage total cases in an area did exist, it was only a slight one. It was decided to evolve the correlation coefficient between these two factors as a test of their relationship. To gain a more accurate idea of the relationship of poverty to case incidence, the correlation coefficient between these two factors was also evolved.

The correlation coefficients were estimated by Mr. Cornish, of the Waite Agricultural Research Institute, and were as follow:

1. That between the density of population and the percentage total cases in an area = 0·61 (significant at P = 0·01).
2. That between poverty and the percentage total cases in an area = − 0·59 (significant at P = 0·01).
3. That between density of population and poverty = − 0·47 (significant at P = 0·05).

The negative signs in 2 and 3 are due to the manner in which the proportion of the population on relief is expressed in the table. The partial correlation coefficient between the percentage total cases and density, after an allowance is made for the effect of poverty, is reduced to 0·47 (significant at P = 0·05). That between the percentage total cases and poverty, after an allowance is made for the effect of density, is reduced to − 0·43, which figure is insignificant.



The questions of the relationship existing in Adelaide between the occurrence of juvenile rheumatism, density of population and poverty, as estimated by their correlation coefficients, may be briefly summarized. Considered by themselves, there is a reasonably close relationship between the occurrence of juvenile rheumatism and both density of population and poverty. However, when the relationship between poverty and density is taken into consideration, the strength of the association is considerably reduced, the correlation between the occurrence of juvenile rheumatism and poverty being in fact reduced to insignificance.

On the basis of these figures for Adelaide it may be said that density of population exerts a greater influence than poverty on the occurrence of the disease. Density of population must, however, be regarded with its associated conditions, such as overcrowding in houses, unhygienic living conditions and the added chances of droplet infection.

Juvenile Rheumatism in Country Areas.

South Australia, 380,070 square miles in extent, presents country areas of varied climates. The west

coast, the upper north and the far north are hot and dry. The mid-north is milder, and some parts of it, such as the Clare district, are damp. The southern portions of the State are colder and wetter; this especially applies to the Adelaide Hills districts and the lower south-east.

In a study of the relative incidence and severity of rheumatic infection in different parts of the State three plans were followed: (i) *questionnaires* were sent to practitioners throughout the State; (ii) an analysis was made of patients with juvenile rheumatism admitted to the Children's Hospital and the Adelaide Hospital from districts outside of Adelaide; (iii) visits were paid to various country towns throughout the State, in which examination of school children and discussion with local practitioners were carried out.

The Reports of Medical Practitioners.

Questionnaires were sent to 232 general practitioners and 125 replies were received. The difficulty in analysing these lay in the fact that the criteria for diagnosis are not clear cut. Frank cases of acute rheumatism present no difficulty, but the much larger group of cases of subacute rheumatism is hard to assess. Furthermore, some practitioners include mild cases of infective polyarthritis or fibrositis in this group, and the labelling of cases of "habit spasm" as chorea is a common diagnostic fallacy.

Although an accurate analysis of the replies received was impossible, certain general impressions were formed:

1. Taking an average throughout the whole of the State, it appears that practitioners encounter 1·0 to 1·5 new cases of acute rheumatism per year in their practices. This figure is practically the same as that given by Boyd Graham[1] for Victoria.
2. For chorea the corresponding figure is somewhat less than 0·5.
3. No impression was formed that acute rheumatism is especially prevalent in any area of the State, except possibly the Clare district.
4. The severity of country cases, compared with those from the city, could not be gauged.

The Hospital Records.

In analysing the records, special note was made of those patients admitted to the hospitals from country districts outside of Adelaide, the place of origin of the attack, and the severity of the disease in each case.

Table II.

Area.	Populated Area in Acres.	Estimated Population (Year 1933).	Total Cases over Eleven-Year Period.	Population per Acre.	Proportion of Population on Relief (Year 1933).	Percentage Total Cases in Area.
City of Adelaide	1,491	29,914	129	20·06	1 in 5·7	0·431
Subdivisions of Adelaide: North Adelaide	528	8,614	23	16·32	Figures not available	0·267
South Adelaide, north of Grote and Wakefield Streets	443	6,200	20	13·19		0·322
South Adelaide, south of Grote and Wakefield Streets	520	15,100	86	29·03		0·569
Enfield	2,400	4,970	20	2·07	1 in 10·4	0·402
Hindmarsh	1,178	13,283	45	11·28	1 in 6·1	0·338
Norwood and Kensington	969	14,524	46	14·99	1 in 7·2	0·316
Campbelltown	3,000	4,262	13	1·42	1 in 22·3	0·305
Port Adelaide	3,550	30,111	75	8·48	1 in 4·6	0·249
Thebarton	1,038	14,756	32	14·22	1 in 9·7	0·216
Marion	1,250	6,526	14	5·22	1 in 7·7	0·214
St. Peters	923	11,735	25	12·71	1 in 11·4	0·213
Woodville	8,300	28,466	58	3·42	1 in 10·5	0·203
West Torrens	4,050	16,979	31	4·19	1 in 11·4	0·182
Colonel Light Gardens	368	4,973	9	13·51	1 in 7·8	0·180
Prospect	1,990	20,663	36	10·38	1 in 15·3	0·174
Henley and Grange	670	4,617	8	6·89	1 in 24·3	0·173
Glenelg	1,093	11,203	15	10·25	1 in 14·1	0·133
Walkerville	600	4,644	6	7·74	1 in 14·4	0·129
Unley	3,515	41,813	57	11·90	1 in 17·2	0·129
Payneham	1,520	8,017	10	5·27	1 in 18·8	0·124
Mitcham	5,350	17,545	20	3·27	1 in 21·7	0·113
Burnside	3,800	22,802	19	6·00	1 in 34·6	0·083
Brighton	1,760	4,885	4	2·77	1 in 18·8	0·081

Conclusions drawn from the results obtained must be very guarded, for there are three obvious fallacies to contend with. First, the number of cases considered is very small. Secondly, it is likely that more patients will be admitted to these hospitals from districts relatively close to the city than those farther away, not only from the fact of the greater distance, but also because there are, in country towns throughout the State, 38 well-conducted Government-subsidized hospitals. Thirdly, in the drawing of any conclusion as to the severity of the disease in a particular district, it has to be borne in mind that in the more severe cases, wherever they occur, the patients are most likely to be sent to Adelaide for the additional observation and treatment that may not be available locally. Thus on the figures alone it would be fallacious to judge that the disease is more severe in one district than another or in the country as opposed to the city. Some useful information, however, was obtained.

Admissions.

The figures given in Table III refer only to those patients admitted to the hospitals in a first attack or to those in whom no cardiac damage had been left by any previous attack. In addition to these 74 patients, five others were admitted. They were all cases of a recurrence of acute rheumatism occurring in a patient with a rheumatic cardiac lesion, the result of a past attack.

TABLE III.

Hospital.	Admissions.	Acute Rheumatism.	Chorea.	Acute Rheumatism Combined with Chorea.
Children's Hospital	42	29	10	3
Adelaide Hospital	32	25	4	3

Places of Origin of Cases.

Patients came from districts well scattered over the State: Ceduna on the west coast, Oodnadatta in the far north, Port Augusta in the upper north at the top of Spencer's Gulf, Berri in the Murray basin, Yankalilla in the south, and Tintinara in the upper south-east. Map III illustrates this.

The fact that no patients were admitted from the lower south-east is almost certainly explained by its distance from Adelaide, together with the presence of hospitals in the area. However, the drier and hotter districts in the State, such as west coast (Ceduna) and upper (Port Augusta) and far northern (Oodnadatta) parts of South Australia, are no bar climatically to the occurrence of the disease. The disease is considerably less prevalent in these hotter, drier areas of the north than in the cooler and wetter districts in the more southern areas, such as the Hills districts in proximity to Adelaide, and the lower south-east. In regard to these Hills districts, of the 79 country admissions no less than 30 were derived from them, and practically all the cases occurred within a radius of sixty miles of Adelaide. The Hills districts are situated in the ranges, running north and south, which are close to Adelaide, and are based on arbitrary divisions used by the Meteorological Bureau. The greater number of the towns shown in the inset enlargement on the map are included in these districts, as are also Mount Pleasant, Woodside, Kuitpo, Willunga, Mount Compass, Yankalilla and Inman Valley, which are shown on the map proper. The districts extend over an area approximately seventy miles long and in places some thirty to forty miles wide, and the altitude of them varies between one and three thousand feet above sea level. The rainfall *per annum* lies between 22 and 46 inches, averaging about 25 to 35 inches, so that during the winter they are the damp areas of the State.

MAP 3
Eyre Peninsula
Spencer's Gulf
Gulf St. Vincent
Kangaroo Id.
New South Wales
Victoria
ENLARGEMENT
MAP of the SOUTHERN PORTION OF SOUTH AUSTRALIA
Places of origin of cases admitted to the Adelaide and Children's Hospitals during 1928—1938 on account of Acute Rheumatism or Chorea.
Note.
One case from each place unless otherwise indicated thus (2)

A fair comparison can be made between the number of patients admitted from these Hills districts and those from the drier areas on the plains north and to a less extent south of Adelaide, as these two parts are approximately the same distance from Adelaide. The former show a far higher incidence of the disease.

The Severity of the Disease.

Although not strictly comparable, the cardiac damage sustained by the 74 patients admitted from country districts can be contrasted with that which

occurred in patients admitted in a first attack from the metropolitan area only. By far the majority of the 74 from the country were also first attack cases, and the few remaining cases were those in which, although a previous attack of juvenile rheumatism had occurred, this had been uncomplicated by any cardiac damage. The comparison between the two groups of cases is a reasonable one.

There was a relatively higher incidence of severe lesions, pericarditis, and combined mitral and aortic valvulitis in the country cases. An appreciably larger percentage of country patients progressed to some cardiac lesion than did corresponding patients from the metropolitan area. For instance, of 42 admitted to the Children's Hospital from the country, 23 (55%) sustained some cardiac damage, whereas only 101 (38%) of 263 admitted from the metropolitan area were so affected.

Two other sets of figures indicate, with the reservations mentioned before, that the disease is of greater severity in country districts than in the city. For, whereas the mortality rate among patients admitted to the hospitals from the metropolitan area was found to be 2·8%, the figure for the corresponding country patients was 5·4%.

Furthermore, nodules are indicative of severity of infection, and in all cases of active rheumatic infection in patients admitted to the hospitals from the metropolitan area they occurred in only 2%, as opposed to the figure of over 7% for the country cases.

What are the reasons for this apparent greater severity of the disease in the country? That in the more severe cases the patients are sent to Adelaide for additional treatment may be a partial explanation. On the other hand, it may be that those in the country, especially children, are more liable to a severe type of infection in accordance with the fact that people in scattered areas develop less herd immunity than those in cities, and that, when infection does occur, it is often of a more virulent type for this reason.

Owing to the small numbers dealt with, it is not possible to single out any particular country district as being that in which the more severe type of infection occurs. However, of areas equidistant from Adelaide, such as the lower north and the Hills districts, the latter appears to be certainly worse as far as cardiac damage is concerned. Of 27 patients with no cardiac lesion admitted to metropolitan hospitals from these districts, permanent cardiac damage occurred in 15, or 55%, which is a high figure when compared with 40%, which refers to all corresponding patients admitted from the other country areas. Furthermore, among the 30 admissions from the Hills districts, three patients showed the presence of nodules and four developed pericarditis, one of these dying in the acute phase of the infection. Of the remaining 49 patients admitted from the country, nodules occurred in only two, pericarditis occurred in two, and there were two deaths.

What are the factors in the Hills districts leading to more severe infection and higher incidence of the disease than in other areas, such as the upper north?

First, the question of rainfall and dampness with cold must be considered. The average annual rainfall for the Hills districts is 25 to 35 inches, which is high when compared with the figures of 9·4 and 13·09 for the upper northern towns of Port Augusta and Quorn respectively. Concerning temperatures, the average maximum summer temperature for Port Augusta is 87·3° F. and the average minimum winter temperature 49·4° F. Contrast figures for Mount Barker, situated in the Hills districts, are 77·2° and 42·6° F.; so that the figures for both these factors of rainfall and cold are quite appreciably lower in the upper northern town of Port Augusta, compared with Mount Barker, which is fairly representative of the Hills district towns.

In the gullies and valleys of the Hills districts there is a greater chance of dampness being present in the homes and wetness in the soil. Fogs are common in the winter months. It appears that hypertrophied tonsils and upper respiratory infections are more commonly met with in the Hills districts than in other areas. Perhaps it is the combined influence of these three factors, namely, cold, dampness and throat infections, that promotes a high incidence of the disease in the Hills districts. In support of this, the high incidence of the disease in the Clare district in the mid-north may be cited. This district has most of the climatic features common to the Hills districts. Thus it is slightly colder in the winter months, the hills among which the district is situated are damp, the annual rainfall is 24 inches, and its altitude (1,300 feet) is very similar to that of many of the Hills districts; also tonsillitis and other upper respiratory infections are very common.

The Examination of School Children in Country Towns.

Acting on the suggestion of the principal medical officer attached to the Education Department, it was arranged that head teachers of the schools visited should look out beforehand for examination those children who (*a*) had had an attack of acute rheumatism or chorea, (*b*) had suffered from "growing pains" or "rheumatism", (*c*) were known or thought to have a cardiac disability of any kind.

Although not ideal, it was felt that few children in the true rheumatic class would be missed by this method, owing to the head teacher's personal knowledge of the illnesses of the children under his charge.

The procedure adopted was to take the medical history and to examine clinically the hearts of the selected children. Naturally it was found that many children singled out for examination did not fall into the true rheumatic class. These were excluded.

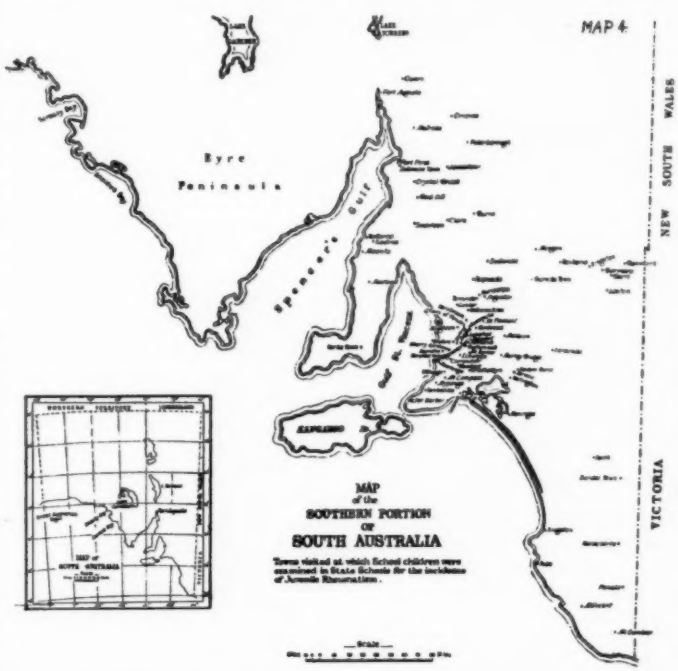
Map IV shows the towns visited. They are situated in six recognized areas: (i) the south-east, (ii) Yorke Peninsula and the mid-north, (iii) the northern area, (iv) the River Murray basin, (v) the

southern districts, (vi) the Hills districts in proximity to Adelaide.

Of the 15,060 children in 95 State schools visited, it was found that 74 had suffered from acute rheumatism and 28 from chorea. In other words, the incidence of juvenile rheumatism amongst the children then on the rolls was 0·67%. This figure refers only to children with a definite history of rheumatic fever or chorea. In a group with a history of "indefinite rheumatism" were included 123 borderline cases. Thus a number who had suffered from subacute rheumatic fever were probably excluded from the true rheumatic class. All children giving a history of "growing pains" were excluded, owing to difficulty in assessing the importance of these.

The above explanation is made, for the figure is very low compared with that given by Kempson Maddox.[15] After perusing some 63,000 medical examination cards of school children in New South Wales, he estimated the general incidence of rheumatism for all rural areas examined at 2·7%.

Regarding the incidence of rheumatic carditis, 26 (0·17%) children were found to present clinical evidence of a rheumatic cardiac lesion. This figure gives only an approximate estimate of the incidence of organic heart disease of rheumatic origin, for it represents the percentage found with this disability in the children who were then attending school. In contrast, Kempson Maddox estimated the rural incidence of rheumatic heart disease in country school children at 0·6%. The difference in the above two sets of figures is probably consequent upon the fact that different systems were adopted in their estimation. In the present investigation all suspected children only were examined, whereas Kempson Maddox derived the incidence of rheumatic infection and carditis from the perusal of school examination cards.



Conclusions.

The information gained from these inquiries is far from precise. More reliance must be placed on that gleaned from personal discussion of the subject with medical practitioners in the various country areas. If the data from all sources are summed up, the impressions as to the varying prevalence of the disease in different parts of the State are as follow:

1. Clare district, situated in the mid-northern area of the State, shows a higher incidence than others.

2. Juvenile rheumatism is rare in the northern parts of the State. Clements[16] mentions, after conducting a nutritional survey among children in the upper north and north-east of the State, that:

> A careful examination was made of all hearts for evidence of rheumatic carditis. Not one case was observed, and further, not one child presented a history of chorea, "growing pains" or rheumatic fever. There were two medical practitioners in the area covered by the survey. In conversation both stated that they had not seen a case of acute rheumatic fever in a child living in the north or north-east of South Australia. It seems reasonable to assume that rheumatic fever does not exist in that country.

Whilst the general truth of this statement may be admitted, the disease is not entirely absent from these parts of South Australia, for in the present study it was found that one patient came from Beltana and another from as far north as Oodnadatta.

3. The disease is relatively rare in the Murray basin towns, where the average annual rainfall is only about 10 inches.

4. Juvenile rheumatism is relatively more common in the following areas: (*a*) the lower south-eastern parts of the State; (*b*) the Hills districts, situated in the Mount Lofty Ranges, in proximity to Adelaide. Although severe cases do occur here, the infection as a whole tends to be fairly mild.

5. An explanation of these impressions seems to lie in the varying climatic conditions existing in the areas. In those areas, the upper north and Murray basin, where the disease has been found to be infrequent, a state of dry heat prevails; but where the disease is more common, as in the Clare district, the Hills districts and the lower south-east, there is the combination of dampness, higher rainfall and relatively cold temperatures.

Home Conditions.

The relationship between home conditions and juvenile rheumatism has been extensively inquired into in England by the Medical Research Council, which in 1927 published a report on "Social Conditions and Acute Rheumatism", showing that

juvenile rheumatism is a disease of the poor class, but not especially of the poorest. In the British Medical Association's inquiry into rheumatic heart disease in children (1926) certain aspects of housing environment, dampness of dwellings, poverty and overcrowding were among the factors considered. Reginald Miller has carried out several investigations into these social factors, and he summarized the above two reports in an article in *The British Medical Journal* on May 28, 1927.

In the present study the home conditions of a group of individuals who had suffered from juvenile rheumatism have been investigated by the almoners attached to the Adelaide Hospital and the Adelaide Children's Hospital. In addition to the inquiries made in Adelaide and suburbs, a small group of cases in the Clare district was investigated, for rheumatic infection was found to be especially prevalent in this area. Table IV indicates the findings.

Table IV.

Observation.	Children's Hospital.	Adelaide Hospital.	Clare.	Total.
Number of homes studied ..	70	40	18	128
Locality of home:				
(a) Poorly drained, or low-lying area ..	14	5	8	27
(b) Residential district:				
(i) Very good ..	5	2	4	11
(ii) Good	40	23	14	77
(iii) Poor	25	15	0	40
State of repair of home:				
(a) Poor	30	11	6	47
(b) Well kept	40	29	12	81
Overcrowding in home ..	16	5	2	23
Dampness in home.. ..	12	6	5	23
Economic class of family:				
(a) Above basic wage ..	6	9	6	21
(b) On basic wage ..	41	7	7	55
(c) Small income below basic wage	7	12	4	23
(d) On relief	16	12	1	29
Diet of patient inadequate..	5	2	0	7
Clothing of patient inadequate	14	2	0	16
Home care and supervision of patient:				
(a) Good	52	30	16	98
(b) Poor	18	10	2	30

Locality of Home.

The residential district was classed as very good if the majority of houses were found to be better class ones and if there were no adjacent thickly populated areas or factories. A good district was one in which most of the residents belonged to the artisan class. In such a district the homes were not crowded together, the great majority of them were of a smaller but well-built type, and the general surroundings were pleasant. In the poor residential districts the homes were usually of a much smaller and inferior type, often semi-detached and in disrepair; the streets were often narrow, and conditions generally were unhygienic.

Only a small proportion of the homes of patients were situated in a poorly drained or low-lying area, except at Clare, where nearly half the homes visited were in damp surroundings.

State of Repair of Homes.

When there was evidence of cracked walls, lack of paint, leaky roofs, or other unhygienic conditions, the home was classed as being in poor repair. It was not found that the type of house or its state of repair had any particular bearing on the disease. The large majority of the homes were clean and well kept. Investigations carried out in Great Britain have shown that juvenile rheumatism shows a relatively high incidence in basement houses in which the factors of dampness, lack of sunshine, and often overcrowding are present. This type of house is practically non-existent in Adelaide.

Overcrowding in the Home.

No precise criteria of overcrowding were laid down, but a general impression was formed, the number of people residing in the home and the number of rooms available for sleeping and other purposes being noted. In only 23 out of the 128 homes was overcrowding present, so this factor was not of significance. Perhaps the main importance in overcrowding is that it predisposes to spread of disease in a household by droplet infection, particularly the common upper respiratory infections, like coryza, mild influenza, naso-pharyngitis and acute tonsillitis. Such infections, when associated with hæmolytic streptococci, are intimately concerned in the ætiology of the disease.

Dampness in the Home.

In only 23 out of the 128 homes was there evidence of damp walls or floors. This factor was not found to be an important one, although it is generally accepted as predisposing to rheumatic infection. One practitioner in a poorer industrial area in Adelaide remarked that the majority of patients suffering from acute or chronic rheumatism met with in his practice came from homes in which there were no efficient fireplaces or other means of producing warmth in the winter. Many of these homes had damp cement floors.

Economic Class of Family.

It was found that 78 out of the 128 patients were derived from families either on the basic wage or on a small income below this, whereas only 29 came from families on relief. As a comparison, the economic class of an unselected group of 500 hospital out-patients, taken in rotation of attendance, was inquired into. The percentages of these and

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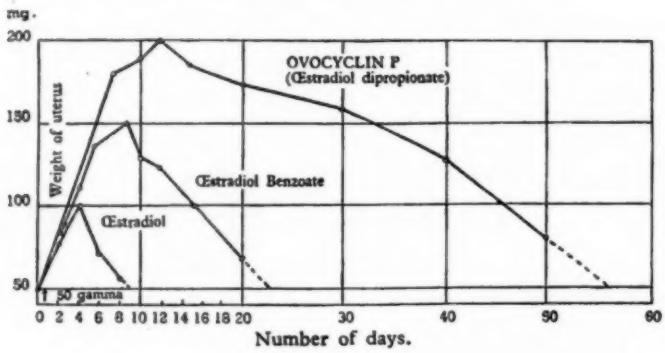

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of the rheumatic group which fell into each class are listed in Table V.

TABLE V.

Economic Class of Family.	Out-patients. (*Per centum.*)	Rheumatic Group. (*Per centum.*)
Above basic wage	7	16
On basic wage	47	43
Small income below basic wage	11	18
On relief	35	23

It is seen that relatively fewer rheumatic patients were derived from families on relief than from the general group of hospital out-patients. Rheumatic infection occurs more often in families in receipt of a small income near the basic wage than in those in receipt of rations. This conforms with the findings of the Medical Research Council in England and with the view of Reginald Miller:[17]

> Rheumatic infection in children shows a very peculiar class incidence. It is very distinctly a disease of the poor . . . yet, although rheumatic infection is thus a disease of the poor, it is hardly a "poverty disease" in the strict sense, as it does not get commoner as the lower depths of poverty are reached. It is not the children of the out-of-work and unemployable that are particularly prone to rheumatism but those of the respectable artisan.

The children of the well-to-do usually escape the disease. Glover,[5] in his Milroy Lectures in 1930, states:

> No disease has a clearer cut social incidence than acute rheumatism, which falls perhaps thirty times as frequently on the poorer children of the industrial town as on the children of the well-to-do.

Practitioners in Adelaide find that juvenile rheumatism is very rare in private practice, compared with its occurrence in the public hospital class. At Saint Peter's College, where there are approximately 160 boarders, the medical officer attached to the school can recollect no case of acute rheumatism in ten years. At Prince Alfred College no case occurred among the boarders in five years.

Diet.

Only broad information on diet was obtained from the parents. In almost all the cases investigated the diet was well balanced and adequate, although a few individual patients lacked sufficient milk or meat or fruit. Under-nutrition does not appear to be a factor of importance in the ætiology of the disease in Adelaide.

Clothing of Patient.

It was judged whether or not the clothes of the patient were adequate for warmth in the winter time. Only 16 out of the 128 patients were found to have clothes that were inadequate.

Home Care and Supervision of Patient.

Impressions were formed as to whether or not the patient was well looked after in its home by its parents. In regard to 98 out of 128 patients this was thought to be satisfactory.

Summary.

In the investigation into the home conditions of 128 patients no outstanding environmental factor was found. Children of families in receipt of a small income but not on relief are somewhat more liable to the disease than others. In some of the cases studied the factors of dampness, overcrowding, lack of home care and unhygienic conditions within the home appeared to be of ætiological importance.

Mortality Statistics.

The difficulty in estimating the incidence of acute rheumatism from death returns is well recognized, and Glover[5] quotes Sir William Church's statement that "it is almost impossible to form any opinion as to the frequency of its occurrence in the United Kingdom".

English and Australian Figures.

Figures were supplied by the Government statist showing the deaths due to rheumatic fever and chorea as a percentage of total deaths. Comparison of figures for England and Wales, as compiled by Glover, with those for South Australia and Australia as a whole is of interest. The South Australian and Commonwealth figures were computed from the returns for 1928-1937 inclusive (see Table VI).

TABLE VI.

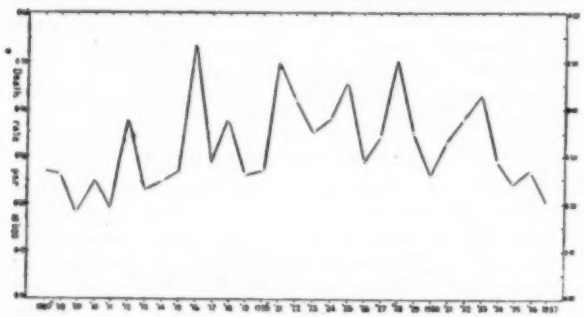
Group.	Deaths.	South Australia.	Commonwealth of Australia.	England and Wales.
1	Percentage of total deaths at all ages from rheumatic fever and chorea	0·38	0·38	0·34
2	Percentage of total deaths at all ages under 15 from rheumatic fever and chorea	0·98	0·89	0·80
3	Percentage of total deaths at ages 10 to 15 from rheumatic fever and chorea	5·25	5·50	5·4
4	Percentage of total deaths at ages 10 to 15 from cardiac disease (all forms). (Glover considers that 80% of the deaths in this group at these ages are due to rheumatic carditis)	6·16	6·73	10·8
5	Percentage of total deaths at ages 10 to 15 from rheumatic fever and chorea and cardiac disease (all forms)	11·41	12·23	16·2
6	Percentage of total deaths at ages 10 to 15 from tuberculosis (all forms)	6·70	5·31	20·7
7	Percentage of total deaths at ages 10 to 15 from scarlet fever	0·54	0·84	7·1
8	Percentage of total deaths at ages 10 to 15 from measles	0·73	0·66	
9	Percentage of total deaths at ages 10 to 15 from diphtheria	2·72	2·42	

Glover especially chose the age period ten to fifteen as being the one in which acute rheumatic infection is most fatal. A striking similarity between the sets of figures in the first three groups is demonstrated, suggesting that, as a cause of death, juvenile rheumatism is as prevalent and important a disease in South Australia and Australia generally as it is in England and Wales. When rheumatic heart disease is taken into account, as in groups 4 and 5, the figures for England and Wales are less favourable, showing the disease to be a greater cause of death at these ages in that country than in Australia and South Australia. An unfavourable climate and the greater degree of industrialization and density of population in England and Wales doubtless exert a strong influence.

Another interesting comparison is between the three sets of figures in groups 5 and 6. In Australian children of ten to fifteen years rheumatic fever, chorea and cardiac disease cause twice as many deaths as tuberculosis causes. In England and Wales tuberculosis was the greater killer of children of those ages.

Falling Death Rate from Rheumatic Infection.

The annual crude death rates per 10,000 population from rheumatic fever in South Australia for the period 1907-1937 inclusive are shown in Graph I.



Graph I.

Illustrating the annual crude death rate per 10,000 of population from rheumatic fever in South Australia over the period 1907-1937 inclusive.

Owing to certain differences in notification of the disease rheumatic fever on death certificates, the figures were strictly comparable only from 1921 onwards. From this year there has been a steady fall in the crude death rate from rheumatic fever, especially since 1928. Glover demonstrated a steady fall in the mortality rates for England and Wales from 1901 to 1928. Atwater[4] noted a similar decline in the United States registration area over the years 1900 to 1923. The comforting fact is that the incidence and/or the severity of the disease in South Australia is on the decline.

In addition to the downward trend of the mortality curve are variations which show a tendency to cyclic recurrence. These occur every three or four years, and in this respect resemble closely the variations in the death rates from rheumatic fever in the United States registration area. Atwater points out, however, that these variations are not peculiar to rheumatic fever, for certain streptococcal diseases, such as scarlet fever, erysipelas and puerperal septicæmia, and some other diseases, such as typhoid fever and dysentery, show a similar three-year to four-year periodicity.

Mortality from Heart Disease.

Table VII shows the death rates in age groups from all forms of heart disease in the years 1911, 1921 and 1933 in South Australia. These were census years. The death rates per 10,000 of population at various age groups were obtained by averaging deaths over five calendar years.

In all the age groups in which rheumatic heart disease is the main factor, there has occurred a fall in deaths from heart disease of all types, again demonstrating the lessening prevalence and/or severity of the infection in South Australia. This fall is particularly evident in the age groups up to twenty-four.

The general increase in deaths from cardiac disease over all ages is explained by the high figures for the age group over sixty-five. In this group the increase in death rate is mainly explained by the fact that there was a far greater proportion of the population existing at advanced ages in 1933 than in 1911. It is also to be noted that since 1930 "senile cardio-sclerosis" or some similar term has largely replaced "senility" on death certificates of elderly people, so producing an increase in the mortality statistics for heart disease.

Table VII.

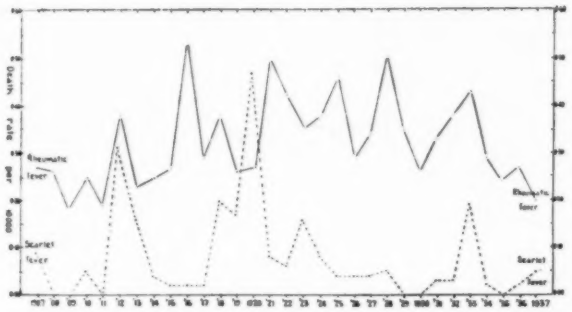
Age Group.	Males.			Females.			Combined.		
	1911	1921	1933	1911	1921	1933	1911	1921	1933
0 to 4	0·10	0·01	0·04	0·13	0·07	0·04	0·11	0·04	0·04
5 to 9	0·10	0·09	0·05	0·08	0·15	0·05	0·09	0·12	0·05
10 to 14	0·16	0·17	0·04	0·11	0·13	0·09	0·14	0·15	0·06
15 to 24	0·21	0·16	0·07	0·25	0·19	0·10	0·23	0·17	0·08
25 to 34	0·26	0·25	0·17	0·34	0·26	0·17	0·30	0·25	0·17
35 to 44	0·77	0·43	0·38	0·64	0·50	0·38	0·71	0·46	0·38
45 to 54	1·27	1·19	1·28	1·31	1·01	0·87	1·29	1·10	1·07
55 to 64	4·12	3·70	4·26	3·21	2·84	2·47	3·69	3·28	3·35
Over 65	12·59	12·36	16·94	12·27	11·07	13·14	12·43	11·71	14·93
All ages	1·14	1·15	1·69	1·10	1·04	1·38	1·12	1·10	1·54

Relationship with Scarlet Fever.

Many investigations have demonstrated an apparent relationship between rheumatic fever and scarlet fever. It has been found that there is a general tendency for bad years for one disease to be bad ones for the other. This has been considered a point in favour of the streptococcal causation of acute rheumatism.

To find out whether such a relationship existed between the two diseases in South Australia, a comparison was made between the annual crude death rates per 10,000 of population for the two diseases over the period 1907 to 1937 inclusive.

This comparison is set out in Graph II. There is a reasonably close synchronism in the oscillations of the two diseases in this State. This relationship is suggestive of some common ætiological factor.



GRAPH II.

Illustrating comparison between the annual crude death rates per 10,000 of population from rheumatic fever and scarlet fever in South Australia over the period 1907-1937 inclusive.

THE INFLUENCE OF SEASON AND CLIMATE.

Wintry Conditions Predispose.

Newsholme[18] and others have stressed the influence of the seasons on the incidence of juvenile rheumatism. In England, autumn and winter are the worst periods; in the United States of America, winter and spring. Everywhere it is least common in summer. Variations in these general trends have been shown by Coburn[6] to reflect the incidence of upper respiratory infections, and Rinehart[19] has noted that peaks of the curves follow periods of relatively little sunshine.

Hospital records show that in South Australia the maximal incidence occurs usually in the winter months. This is not always so, for in 1929 the highest incidence was in January, the middle of summer. Graph III shows the pooled monthly figures for the period 1928 to 1938. The disease is least prevalent at the end of spring and in the summer; it is commonest in winter, reaching its maximum in July.

Rainfall and Temperature.

Many authorities regard these climatic factors as important. Poynton and Schlesinger[20] consider that sudden atmospheric changes to damp cold after a long spell of dry, hot and dusty weather are likely to lead to the first attack.

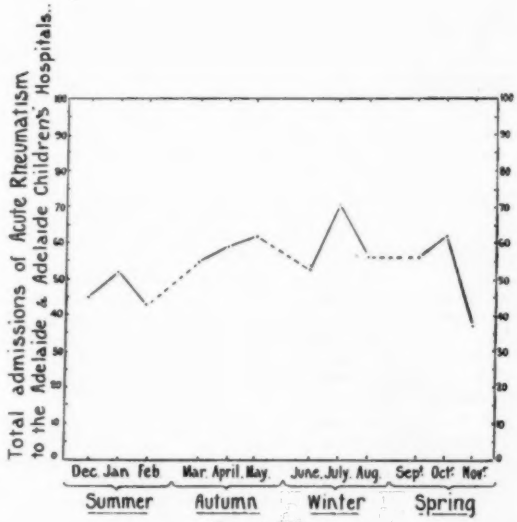
The figures for hospital admissions for acute rheumatism were compared with variations in rainfall and temperature in each month. High rainfall periods generally gave more cases, but low temperatures appear to have little influence on incidence.

The test of the correlation coefficient was applied to the factors of rainfall and temperature against admissions to hospital in months. These coefficients were estimated by Mr. Cornish, of the Waite Agricultural Research Institute. He summarizes the matter:

> A regression analysis of the effect of temperature and rainfall of the current and previous months on the admissions for acute rheumatism to the two hospitals disclosed no significant relationships. The partial regression coefficients on admissions of temperature, however, did tend to negative values. In other words, low temperatures had a tendency to be associated with an increase in the number of admissions. The regressions on rainfall were in general positive, particularly for the winter months, a greater number of admissions tending to be associated with a higher rainfall.

The analysis was carried out in months, and it is pointed out that the results were obtained from the consideration of a relatively small number of years.

The results of the estimation of these correlation coefficients disclosed that there was a general trend for the wetter months, with colder temperatures, to be associated with an increase in the number of rheumatism admissions to hospital. However, no significant relationship was definitely established from the data analysed.



GRAPH III.

Illustrating seasonal incidence of acute rheumatism in South Australia over the period of eleven years, 1928-1938 inclusive.

EXPERIMENTAL AND LABORATORY STUDIES.

As part of the research, some laboratory studies were carried out at the Institute of Medical and Veterinary Science. Professor Hurst directed the experimental work on monkeys, and himself per-

formed most of it. Professor Platt supervised the bacteriological work. The material was collected by the research officer, and the institute staff performed most of the detail work. The laboratory inquiries were more restricted than intended, for it so happened that the period of the inquiry, the year 1938 and the first half of 1939, was marked by a low incidence of the disease, and suitable material from patients was scarce.

Examination of Joint Effusions.

In only six of the 105 patients coming under observation during the research was synovial effusion sufficiently great to allow of aspiration. The fluid obtained was immediately mixed with sterile citrated saline solution. A direct smear was made. The fluid was incubated aerobically in serum and on blood agar plates, and anaerobically, the same media and Robertson's cooked meat medium being used. The incubated media were observed for five days. The findings here recorded are summarized from the official reports of the institute. The results were:

Case I.—A man, aged twenty-nine years, suffered from acute rheumatism with polyarthritis. The onset was preceded by trauma to a knee. The fluid obtained was yellow and rather viscid, and macroscopically clear. A direct smear contained numerous polymorphonuclear leucocytes; no organisms were found. Aerobic culture gave growth of a diphtheroid organism, and anaerobic culture one of *Streptococcus fæcalis*; both organisms were regarded as contaminants.

Case II.—A boy, aged seven years, suffered from acute rheumatism with fluid in both knee joints. The fluid was yellow, viscid and turbid. A direct smear contained a large number of cells, all polymorphonuclear leucocytes. A few Gram-positive cocci were present. No growth resulted from attempted culture.

Case III.—A man, aged sixty-one years, suffered from acute rheumatism with pericarditis. He gave a history of acute rheumatism in youth. The relapse was preceded by acute tonsillitis three weeks previously. The joint fluid was clear and yellow. Numerous cells were seen in the direct smear, most of them polymorphonuclear leucocytes. Gram-positive diplococci were present. Culture was a pure growth of a Gram-positive spore-bearing micro-aerophile organism, requiring enriched medium for its growth. A culture of the organism was injected intramuscularly into a guinea-pig without result.

Case IV.—A boy, aged sixteen years, suffered from acute rheumatism. Joint fluid was yellow and turbid. A direct smear showed numerous cells, mostly polymorphonuclear leucocytes; Gram-positive diplococci and rods were present. An organism was obtained in pure culture; it was a Gram-positive spore-bearing micro-aerophile, requiring enriched medium for its growth.

Case V.—A girl, aged ten years, suffered from severe acute rheumatism with pericarditis. The joint fluid was yellow and turbid. A direct smear contained no organisms; but numerous polymorphonuclear leucocytes were present on culture; an organism similar to that from Case IV was produced. On blood plates two small greyish colonies developed after three days' incubation. The organism was a Gram-positive bacillus. It failed to grow in a subculture of serum broth incubated anaerobically for four days.

Case VI.—A boy, aged sixteen years, suffered from acute rheumatism with polyarthritis. The joint fluid was yellowish and blood stained, probably from trauma of aspiration. A direct smear contained numerous erythrocytes and a few polymorphonuclear leucocytes, but no organisms. No growth resulted from attempted culture.

Professor Platt reported on the findings:

The organism obtained from Cases III, IV and V was a Gram-positive spore-bearing bacillus. It was recovered by anaerobic culture. Upon isolation the organism grew very poorly on enriched laboratory media, giving small semi-translucent mucoid colonies on blood plates. With subculturing the organism adapted itself to growth on ordinary heart extract agar. The organism was an active carbohydrate fermenter, and attacked a wide range of mono-, di- and poly-saccharides, with the production of acid and gas. The organisms obtained by culture from the three cases died off rapidly, with the exception of the one isolated from Case III, the growth of which could be retained in subsequent subcultures.

The results of inoculation into monkeys of joint fluids from four of the above patients were as follow:

Case II.—Fluid was inoculated intratesticularly and intramuscularly into two monkeys (*Macaca mulatta* and *Pithecus entellus*) without any result during one month's observation.

Case III.—Fluid was inoculated intratesticularly and intramuscularly into a monkey (*Macaca irus*) without effect during an observation period of four weeks.

Case IV.—Inoculation was made intracerebrally and intraarticularly into a monkey (*Macaca maurus*). The animal died on the sixth day after inoculation, and without showing pyrexia. Culture of the brain yielded the same organism. Section of brain tissue showed only minimal inflammatory lesions at the site of inoculation and elsewhere.

Case V.—Fluid was inoculated intracerebrally, intramuscularly and intraperitoneally into two monkeys (*Cebus fatuellus* and *Macaca radiatus*) without producing any reaction in the four-week period of observation.

From these investigations it is not possible to infer that the organism, similar in its general characteristics, obtained from these three separate cases of acute rheumatism, is of any ætiological significance. However, its presence in three separate cases is of interest.

Examination of Rheumatic Nodules.

The relative infrequency of nodules in the cases studied has been discussed earlier in this report. In the patients observed during the research nodules were noted in only four, and in only two were they of sufficient size to warrant excision for examination. The photomicrograph (Figure I) was taken from one of the sections, and the report attached was supplied by Dr. J. B. Thiersch, Neale research pathologist at the institute. In each case suspensions of nodules in saline solution were injected into monkeys. In one the suspension was injected intracerebrally into the monkey (*Macaca nemestrinus*), but produced no pyrexia or other sign in twenty-eight days. In the other case the injection was made intracerebrally and intratesticularly into a monkey (*Macaca radiatus*) with no effect in forty days.

Throat Swabs.

It was considered beyond the scope of this research to undertake a large-scale programme of regular swabbing of the throats of a series of patients, to study any changes in the bacterial flora over a period of several months. To obtain some information about the presence and types of strepto-

ILLUSTRATIONS TO THE REPORT ON RHEUMATIC INFECTION IN SOUTH AUSTRALIA.

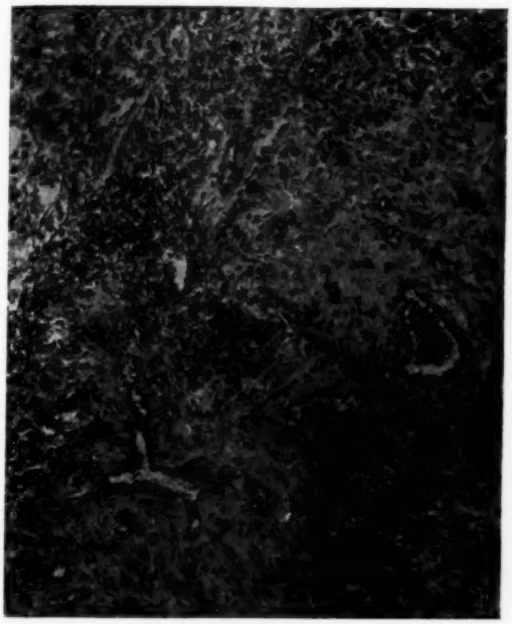


FIGURE I.
The section of a rheumatic nodule. The nodule consists of granulomatous tissue formed by proliferation of dark nucleated spindle cells of fibroblastic and histiocytic origin with polymorphic and eosinophilic infiltrations. The connective tissue is swollen, gelatinous and in parts necrotic. There are marked swelling and œdema of the adventitial tissue of smaller vessels and periadventitial infiltrations. There are also multinucleated giant cells of rheumatic type.

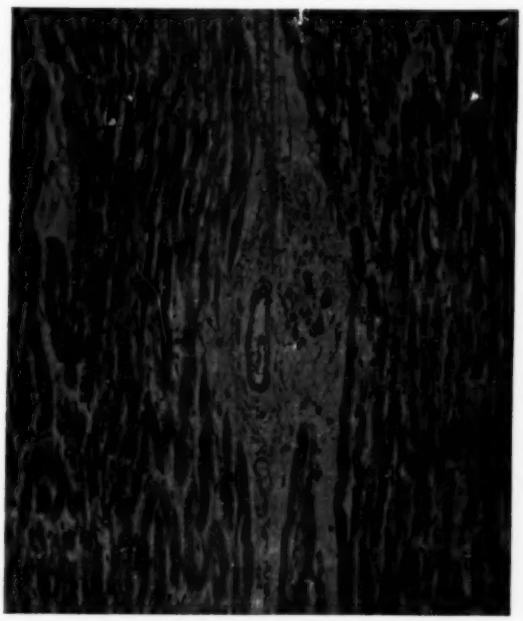


FIGURE II.
Aschoff body in the periadventitial connective tissue of a branch of a coronary artery. Note the dark, multinucleated giant cells and the swelling of the connective tissue.

ILLUSTRATIONS TO THE ARTICLE BY DR. G. M. OXER.

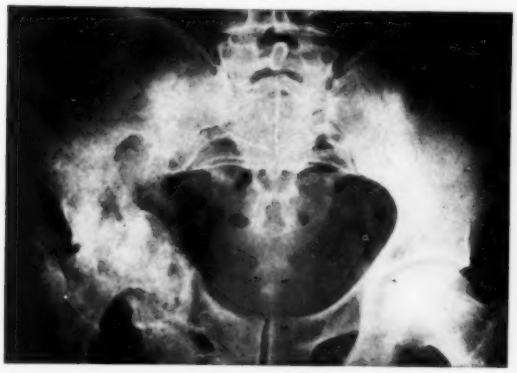


FIGURE I.
Skiagram of pelvis 22 months before death.

FIGURE II.
Skiagram of pelvis six weeks before death.

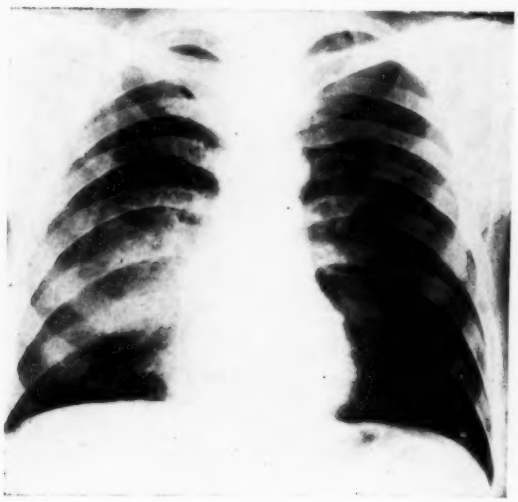


FIGURE III.
Skiagram of the lungs five weeks before death.

ILLUSTRATIONS TO THE ARTICLE BY Dr. DOUGLAS ANDERSON AND Dr. R. A. MONEY.

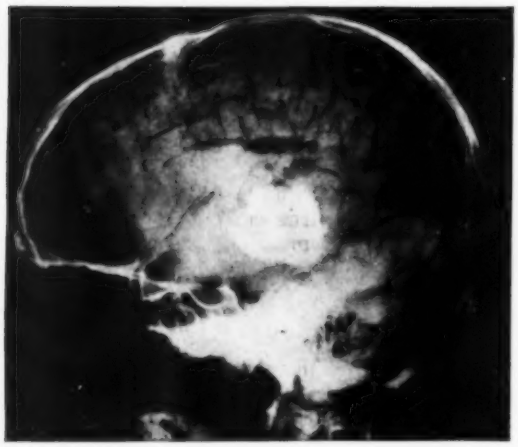


FIGURE I.

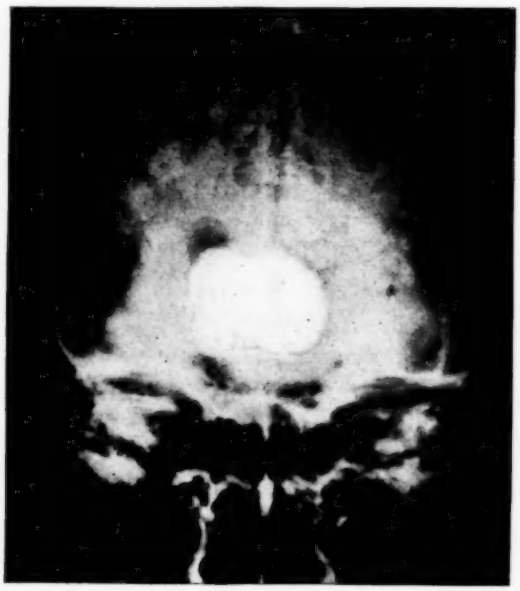


FIGURE II.

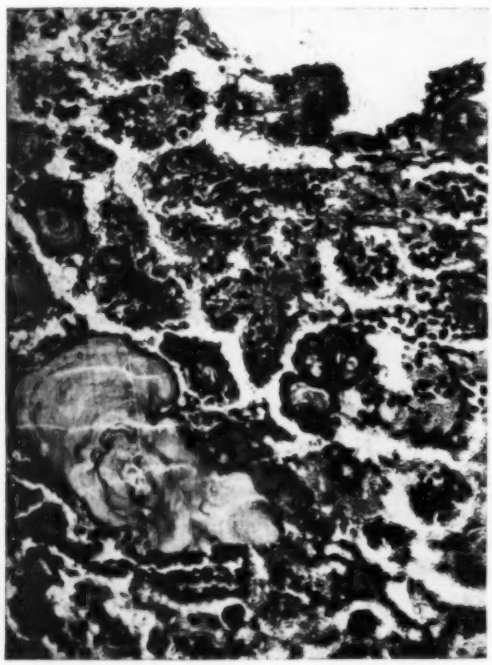


FIGURE IV.

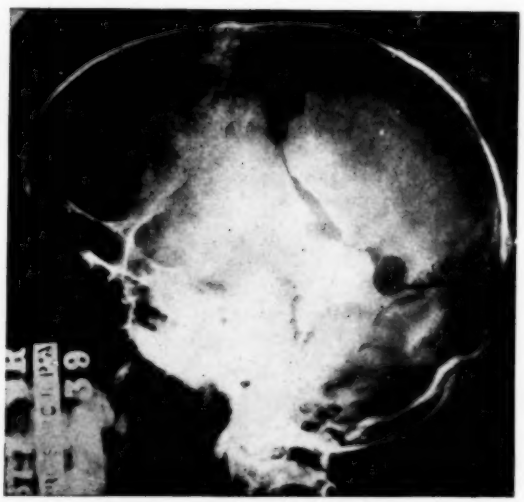


FIGURE VII.

cocci in the throats of rheumatic and non-rheumatic children a small group of 60 was investigated by means of swabs taken from the surface of the tonsils, with the results set out in Table VIII.

Table VIII.

Group of Children.	Number of Children.	Pneumococci or *Streptococcus viridans*.	Hæmolytic Streptococci.	Inert Streptococci.
		%	%	%
Non-rheumatic children from the general Medical Out-Patients' Department at the Children's Hospital	20	85·0	35·0	25·0
Non-rheumatic children in medical wards	18	77·7	50·0	5·5
Convalescent rheumatic children	22	95·4	63·6	27·2

It is generally recognized that an attack of acute tonsillitis, or even of mild naso-pharyngitis, may be followed after an interval by one of acute rheumatism. In such cases hæmolytic streptococci may be found in the throat swab taken during the initial illness, but they commonly disappear from the throat flora in a few days and before the onset of the rheumatic attack. Swabs taken when the patient has been admitted to hospital are therefore not a true indication of whether infection with hæmolytic streptococci has initiated the rheumatic attack. Of forty patients whose throats were swabbed within a few hours of admission to hospital, hæmolytic streptococci were found in 10, in 25 there were non-hæmolytic streptococci, and in the remaining five cases no streptococci were found.

Scurvy and Acute Rheumatism.

Rinehart and Mettier[21] produced in guinea-pigs lesions similar to those found in acute rheumatism in human subjects. The animals were first made scorbutic and then inoculated intracutaneously with hæmolytic streptococci. In the present work the attempt was made to study this matter further; monkeys were used for the experiment. In such experiments it is desirable to use an organism which is normally pathogenic to the animal; but as no strain of hæmolytic streptococci known to be pathogenic to monkeys was available, the hæmolytic streptococci obtained from a human case of acute follicular tonsillitis were used in this work.

Six rhesus monkeys were placed on a scurvy-producing diet for a little over two months, by which time the animals were in a severely scorbutic state. Normally fed monkeys were used as controls. Approximately 25,000 hæmolytic streptococci were then injected into the tonsils of one of the scorbutic and one of the control animals. Two corresponding animals had their tonsillar fossæ swabbed with a broth suspension of this organism. No rises of temperature or other reactions were obtained in any of the animals over a period of one month's observation. During this time the scorbutic animals were kept on their scurvy-producing diet. A similarly planned experiment was done, a strain of hæmolytic streptococci obtained from the throat of a healthy monkey being used. No reactions occurred in the observation period of one month.

Summary of Laboratory Findings.

It is acknowledged that the scope and amount of the laboratory studies were limited, but the results may serve as a guide for further investigations:

1. Organisms having the same general characters were obtained from joint fluids of three patients with acute rheumatism. The significance of this finding is not established.

2. No significant results were obtained from the inoculation of monkeys with material from active lesions of patients suffering from acute rheumatism, nor in guinea-pigs from the inoculation with cultures of organisms from joint fluid. The search for evidence of a virus capable of transmission to an animal and of producing lesions identical or resembling those of juvenile rheumatism in the human subject has yielded negative results.

3. No reactions occurred in scorbutic monkeys after tonsillar injection with cultures of two strains of hæmolytic streptococci, one of which was of monkey origin.

4. Hæmolytic streptococci were detected in the throats of a higher percentage of convalescent rheumatic children than in a control series of non-rheumatic out-patient children. When compared with non-rheumatic in-patients, the greater incidence in the convalescent rheumatic subjects was not so marked. The series so studied was too small to enable the drawing of sound conclusions.

Rheumatic Infection in Aborigines.

The Mission Stations.

There are in South Australia two mission stations for the care of aborigines. One is at Point Pearce on Yorke Peninsula; the other is at Point McLeay, on the edge of Lake Alexandrina, some ninety miles south-east of Adelaide. These were the only aboriginal communities easily accessible for inquiries, and visits were made to each.

There were extremely few full-blood natives living at the mission stations. At Point Pearce there were only five amongst the 330 inhabitants, and at Point McLeay, where some 420 natives live, the actual number was uncertain, although it was stated to be only six or ten. The majority of the natives in the missions are half-castes or quarter-castes, and in several of these juvenile rheumatism was found to have occurred.

It was not possible in the time available to examine all the inhabitants of the stations for evidence of past rheumatic infection. The following reports relate to those natives known by the sister or visiting medical officer to have suffered from a form of juvenile rheumatism.

Point Pearce Mission Station.

Case I.—A half-caste married woman, aged twenty-three years, had been born at the mission and lived there all her life except for two or three short holidays. At the

age of twelve she had a typical attack of acute rheumatism. During each of her three pregnancies and often with cold changes of weather she suffered from exacerbations of rheumatic pains in her joints, especially the knees. She suffered from occasional attacks of "sore throat". Her tonsils were not infected and there was no deformity of her joints. Heart examination revealed an apical systolic bruit, conducted out into the axilla.

CASE II.—A half-caste girl, aged twelve years, had been born at the mission station and lived all her life there. From the age of nine she had suffered from intermittent pains in the joints of her arms and legs, occasionally with some swelling. On two occasions she had had to be kept in bed for a few weeks with these symptoms, and several times she had noticed a red blotchy rash present on her legs when the joint pains were present. She did not suffer from sore throats. Examination showed evidence of mitral disease.

CASE III.—A half-caste girl, aged ten years, a sister of the previous girl, was born in Adelaide and taken to the mission soon afterwards. She had not left the mission from that date. She had suffered from attacks of mild joint pains from the age of nine, with very little swelling, and not of sufficient severity to confine her to bed. There was evidence of chronic infection in her tonsils, but she had never had an attack of acute tonsillitis. There was no cardiac abnormality found.

CASE IV.—A half-caste girl, aged twelve years, had been born on the mission station and lived all her life there. During the last eighteen months she had had three or four attacks of chorea. When examined she showed involuntary, purposeless and non-repetitive movements, a state she had been in for the three weeks prior to the visit. On examination of the heart, the first sound at the mitral area was roughened and accentuated, but no definite bruit was present, either at rest or after exercise.

Point McLeay Mission Station.

CASE I.—A half-caste girl, aged seventeen years, in 1938 spent the greater part of five months in bed with fairly severe chorea, and was attended by the visiting medical officer. The physical examination disclosed no organic lesion of the heart and no tonsillar infection.

CASE II.—A half-caste boy, aged twelve years, during the last few winters had had "rheumatic pains" in both knee joints, sufficiently severe to keep him in bed for a week at a time. No abnormality was detected in the heart.

CASE III.—A quarter-caste girl, aged nineteen years, two years ago, about ten days after an attack of acute tonsillitis, developed a migratory polyarthritis, with much pain and very little swelling of the joints. With this illness she was in bed for twelve months. The illness was diagnosed as severe acute rheumatism by the visiting medical officer, who at the time considered her heart to be affected. Examination later showed no cardiac abnormality, and there was no evidence of chronic tonsillar infection.

Reports of Other Observers.

The chief quarantine officer at Darwin, replying to inquiry, stated:

There is no record here of any rheumatic fever amongst aborigines, nor have the medical officers any knowledge of this condition having occurred in these people.

Cleland[22] reports:

Dr. Gorrie, Port Augusta, states that rheumatic fever is common among aboriginals. Cardiac trouble is also quite common, and post-mortem examinations show typical changes affecting the valves and endocardium. Dr. Illingworth (Taroom, Q.) has met with three cases of rheumatism, one each in the knee, shoulder and hip. He has met with no acute cases of rheumatic fever, but has had one example of mitral stenosis, which was post-rheumatic.

Cleland[23] also recorded the *post mortem* findings in the case of a full-blood aboriginal, aged thirty-six years, who had had rheumatic fever twenty-two years before death. Cleland found a typical post-rheumatic mitral valve lesion with recent vegetations, and, in a personal communication, has stated that it was the only *post mortem* example he could recollect having seen of a rheumatic valvular lesion in a full-blooded native.

At the Children's Hospital in 1937 a half-caste aboriginal boy (Australian aboriginal mother, Fijian father), aged three and a half years, died from rheumatic carditis. Three months before admission he had been treated as an in-patient for acute streptococcal tonsillitis. He recovered and remained in good health for about a month. He then began to sleep poorly, to lose his appetite and to develop pains in his legs. When examined by a doctor at a baby health centre he showed signs of pericarditis. His temperature was normal on admission and respiration was distressed. The cardiac impulse was in the fifth intercostal space but displaced outwards; there was a loud systolic bruit and thrill over the mitral area. No abnormality was detected in any joint. He became rapidly worse and he died thirteen days after admission. *Post mortem* examination showed a small patch of fibrin on the surface of the auricle, and rheumatic vegetations were present on the mitral and aortic valve cusps.

The clinical course of the disease in half-castes and quarter-castes is similar to that in whites. Concerning the reactions to disease in the full-blood, the opinion of Cleland[22] is quoted:

It seems probable that the native (meaning full-blood) suffers from the same diseases as the white man, and in general in much the same way. In spite of his extremely long separation from other races and the purity of his descent, the reactions to disease seem not to have appreciably departed from the types met with amongst other races . . . We may infer that biologically the reactions to disease are essentially primitive and archaic and are likely to be very similar in widely separated species of mammals.

CONCLUDING COMMENTARY.

Preventible Crippling.

Rheumatic infection is a serious matter. The amount of heart crippling it produces makes it a subject of vital importance, even more important than poliomyelitis and its crippling of limbs.

Australia is fortunate in being less severely attacked than most countries; yet even here rheumatic infection and the associated heart disease exact a dreadful toll. To cure the damaged hearts is not possible; to prevent the damage from rheumatic infection is a public health problem worthy of the best efforts. There are far too many heart cripples, and vigorous action is needed to reduce the incidence.

Epidemiological Factors in South Australia.

This problem affects South Australia no more seriously than it does Australia as a whole. Yet, since incidence and severity of rheumatic infection vary in different places, the study of local aspects of the problem should be prosecuted everywhere. The Central Board of Health therefore undertook this investigation to ascertain the local status of the problem, the causative factors and any practicable means likely to reduce the damage.

The research has shown that the predisposing causes are essentially the same here as elsewhere; poverty, damp living conditions and overcrowding play strong parts. Agreement on the nature of the actual cause of juvenile rheumatism is yet to be reached. The streptococci are intimately concerned; yet some features of the illness are difficult to explain on the basis of streptococcal infection alone. Whether an associated virus is present is one of the many problems awaiting solution.

Lines of Attack.

Some health authorities abroad have introduced compulsory notification of acute rheumatism to assist them in assessing the problem and in trying to cope with it. For several years this procedure has been in force in the Royal Borough of Kensington (London), and it has served at least to focus attention on the menace of rheumatic heart disease. There are inherent difficulties in assessing accurately the incidence of rheumatic infection in a community. Although typical rheumatic fever is a clear-cut clinical entity, the less acute forms of rheumatic infection are not. It often happens that scant respect is paid to the minor types of rheumatic infection. It cannot be stressed too strongly that these minor infections are as grave a danger as frank cases of rheumatic fever, for they may cause irreparable and disabling cardiac complications. With the types of illness often ill-defined and the actual cause still unknown, compulsory notification is necessarily limited in its value.

In the present state of knowledge the best method of dealing with cardiac crippling is to prevent it. The care of rheumatic patients in the acute phase of their illness and suitable management in convalescence are essential and require ample and good institutional facilities. The establishment of rheumatic supervisory centres in many cities has proved of great value. The rheumatic supervisory centre conducted at the Adelaide Children's Hospital, linked with the arrangement for convalescent treatment at Estcourt House (at the seaside and about eight miles from Adelaide), is an ideal plan, and although this local scheme is a small one it is doing excellent work.

The suitable occupational training of young adults with damaged hearts must not be overlooked. A child whose physical state requires that he rest in an institution for perhaps a year or two may find himself at the end of that period poorly fitted to take his former place in life. After a month or two of the acute phase of the illness has passed, the patient is usually able to undertake some form of study and perhaps a limited amount of manual work. Hospital schools and vocational training centres enable the conduct of some suitable and useful work during the long period of convalescence; neglect of this need may leave the patient a misfit for life. Most cardiac cripples may be suitably trained to do useful work and perhaps earn a livelihood if the opportunity for training is seized at the right time and in the proper manner.

There is still need for continued research into the prime causes of rheumatic infection. Further studies on the matter following the lines of experimental and laboratory studies outlined in Section VIII of this report may eventually lead to valuable additional knowledge. Continued attention to town planning, to sanitation and housing, to health education, to any means for enabling communities to live hygienically, will achieve much. To try to cope more effectively with rheumatic infection—here lies a duty and opportunity for preventive medicine. If this report does something to gain more interest in the problem, and to point out ways for attacking it, the work it records will not have been futile.

Appendix I.

The Significance of Rheumatic Nodules.

Pathologically, the hall-mark of acute rheumatism is the Aschoff body. Figure II is a photomicrograph of an Aschoff body in the myocardium of a patient who died from acute rheumatism with pericarditis. The photograph was prepared and the report supplied by Dr. J. B. Thiersch.

Aschoff bodies most commonly occur between the muscle bundles of the myocardium, in the synovial membrane of joints, in the fibrous tissue of fascial structures, muscles and tendons, and sometimes in the lung.

The structure of a rheumatic nodule is essentially that of an aggregation of separate Aschoff bodies. They usually occur in patients with a severe widespread infection, and especially when this results in gross cardiac damage. Although rather too pessimistic, Cheadle's dictum that "the appearance of massive nodules was the equivalent of a sentence of death" emphasizes their prognostic importance. Their site of election is over bony prominences and insertions of tendons, and the symmetry of their location is often noted. Figures III, IV and V are photographs of nodules in patients at the Children's Hospital.

Figure III.

Showing symmetrical distribution of rheumatic nodules over the olecranon and both epicondyles of the elbows.

Rheumatic nodules may be quite obvious, standing out as rounded prominences under the skin over the knees, ankles, elbows, scalp and scapulæ *et cetera*. Sometimes diligent search by palpation has to be made to locate them. Extreme flexion of the knee, elbow, wrist and ankle joints often makes possible the palpation of a nodule which would otherwise be hidden by the adjacent structures.

It is stated in the report that nodules were present in 5·8% of patients admitted to the Children's Hospital with acute rheumatism. Kempson Maddox[13] found that of 428 rheumatic patients admitted during a period of ten years to the Royal Alexandra Hospital for Children in Sydney, nodules occurred in only 1·5%. He mentions that Bertram found nodules in 7% of patients in Glasgow, Poynton in London from 19% to 28%, and Wallace in 12% of 124 Chicago children.

There was a high fatality rate among the 20 patients admitted to the Children's Hospital, who exhibited nodules; four of them died. The case records showed that the severe lesions of pericarditis and combined mitral and aortic valvulitis were more prone to occur in patients who exhibited nodules than in those who did not, and

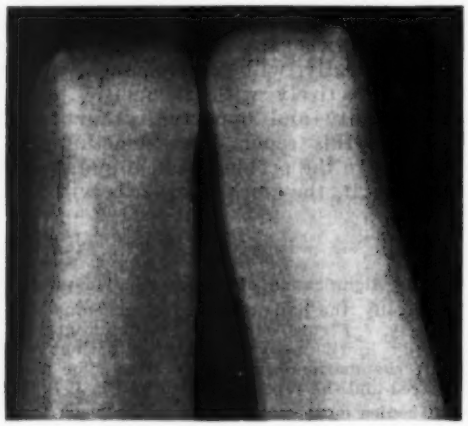


Figure IV.
Showing symmetrical distribution of rheumatic nodules over the lateral condyles of both knees.

that every patient in whom nodules occurred sustained cardiac damage of some degree.

This finding agrees with the view of Schlesinger:[24]

> The almost invariable association of nodules with carditis is common knowledge . . . It is improbable that a rheumatic infection, severe and extensive enough to produce subcutaneous nodules, will ever leave the internal organs, and particularly the heart, free from similar inflammatory lesions. It is almost impossible for nodules to exist without heart disease.

Figure V.
Showing rheumatic nodules situated over the external and internal malleoli of the ankles.

Appendix II.

Skin Rashes in Acute Rheumatism.

Various lesions of the skin may occur in acute rheumatism. They include: (*a*) *erythema marginatum*, (*b*) *erythema multiforme*, (*c*) petechiæ, (*d*) urticaria-like rashes, (*e*) occasionally *erythema nodosum*.

Erythema marginatum is the most typically rheumatic of these skin lesions, and is very distinctive when it occurs. The erythema tends to occur in pink circular patches, with crescentic outlines, and large areas of the skin may be involved at the same time. It is migratory and evanescent in character, and its occurrence in a

Figure VI.
Showing *erythema marginatum* in a patient with acute rheumatism. The lesions are distributed over the arms, buttock, chest and abdomen as small areas, some of which have coalesced, with an irregularly marginate outline.

patient is frequently missed. Figures VI and VII illustrate *erythema marginatum* occurring in a rheumatism patient at the Children's Hospital. The occurrence of *erythema marginatum* is regarded by some as an allergic

Figure VII.
Showing *erythema marginatum* in a patient with acute rheumatism. The lesions are distributed over the arms, buttock, chest and abdomen as small areas, some of which have coalesced, with an irregularly marginate outline.

response to the products of hæmolytic streptococci. Skin tests on individuals who have had acute rheumatism, when the endotoxin fraction of hæmolytic streptococci is used, have been carried out by several investigators, including Coburn,[49] and Collis, Sheldon and Hill.[25] The latter state that "rheumatic children show a much greater response to hæmolytic streptococcal extract than other children, and that the recentness of the last attack of acute rheumatism is an important factor in the production of the reaction".

In many of these skin tests the lesions resembled those met with in acute rheumatism, typical examples of *erythema marginatum* being produced, although in some cases the lesions were so acute as to form hæmorrhagic bullæ or purpuric-like eruptions.

In 24, or 5·3%, of 450 rheumatic patients admitted to the Children's Hospital, some rheumatic skin manifestation occurred during the active phase of the infection; 18 were cases of acute rheumatism. It was not possible, from study of the case records, accurately to classify the types of lesion. The majority were *erythema marginatum*, *erythema multiforme* or some urticaria-like rash.

APPENDIX III.

The Distribution of Movements in Chorea.

Unilateral or hemichorea of the Sydenham's type is a well-known clinical entity. Often the movements are unilateral at the onset, and later become bilateral. Sometimes they are restricted to one side of the body throughout the illness. Macintosh and Anderson[29] record the denial of many observers that a true hemichorea exists, maintaining that slight movements are always to be seen on the opposite side of the body if looked for.

The case records gave the following figures for the Children's Hospital and Adelaide Hospital cases combined:

Number of patients admitted with chorea	223
Bilateral movements	137 or 61·4%
Predominantly right-sided	40 or 17·9%
Predominantly left-sided	23 or 10·3%
Exclusively right-sided	18 or 8·1%
Exclusively left-sided	5 or 2·3%

It is seen that the movements were predominantly or exclusively right-sided in 26·0% of cases and left-sided in only 12·6%.

Osler's figures show that the movements begin most frequently on the right side, whereas Gowers records practically no difference. If the movements in chorea do in fact tend to occur more often on the right side than the left, the explanation is not evident, unless it be related in some way to the predominance of right-handedness.

APPENDIX IV.

The Colouring of Children in Juvenile Rheumatism.

It has often been stated that individuals who suffer from juvenile rheumatism tend to have certain distinctive physical characteristics. Hawksley[27] found that so-called rheumatic "growing pains" occurred more frequently in children with brown eyes and various shades of brown and black hair, while Matthew Young,[28] in an analysis of over 1,000 children, came to the conclusion that no type of child should especially be regarded as "rheumatic". The discussion has revolved mainly around the combination of hair and eye colour and such characteristics as the texture and pigmentation of the skin.

Three groups of children have at one time or another been thought to be the "rheumatic type". They are: (*a*) the blonde child with blue eyes; (*b*) the red-haired and freckled child; (*c*) the brunette, with dark hair and eyes, long eyelashes and a fine-textured white skin. In these children the sclerotics may have a bluish tint and the upper central incisor teeth are often massive. Hutchison[29] places the typically rheumatic child in this third group, while Still regards red-haired children as more prone.

In this part of the study a group of 100 rheumatic children was compared with an equal number of non-rheumatic children. Sources of error were evident, for colour decisions vary with different observers. The findings in the two groups of children are here presented (Table IX).

The grouping of colour combinations was more difficult. The only satisfactory plan was to divide them into those with frankly red hair, those with the fair hair and blue eyes combination, and brunettes. The combination of fair hair and brown eyes is unusual. For the three subdivisions the percentage distribution was as shown in Table X.

TABLE IX.

Colour of Eyes.

Colour.	Control Group.	Rheumatic Children.
Dark blue	6	15
Light blue or grey blue	49	20
Dark brown	11	25
Light brown	11	5
Brown-green (hazel)	17	19
Green-grey	6	16

Colour of Hair.

Colour.	Control Group.	Rheumatic Children.
Blonde	31	13
Dark brown	16	34
Medium or light brown	35	25
Black	3	11
Red	3	4
Nondescript light-coloured	12	13

TABLE X.

Colour Combination.	Control Group.	Rheumatic Children.
Red hair	3	4
Fair hair with blue eyes	36	12
Brunettes	56	82

It is seen from the tables that dark brown eyes or dark brown hair separately were found to occur more than twice as commonly amongst the rheumatic children as amongst the controls. Dealing with the colour combination, the blonde type with fair hair and blue eyes was found to occur three times as commonly among the controls as among the rheumatic group, whereas brunettes were much commoner in the rheumatic children than in the control group.

APPENDIX V.

The Family History in Juvenile Rheumatism.

Much debate has centred around the family history in juvenile rheumatism. Statistics relating to it vary considerably, and investigators differ as to its significance.

A hundred children known to have suffered from juvenile rheumatism were investigated, together with a control group of 100 non-rheumatic children. In each case the child and one of its parents were interrogated. Attention was paid only to the "immediate" family history, that is, a history of juvenile rheumatism in one or other of the parents of the child, or in any brother or sister. In the control group a family history of juvenile rheumatism was obtained in eight of the series, while in the rheumatic children 30 showed rheumatism in the family.

These figures, although obtained from a small group of children, may be compared with those of Read, Ciocco and Taussig.[30] They found that 30·8% of parents of the rheumatic patients whom they investigated showed some rheumatic manifestation, in contrast to 7·7% among the parents of their control group, that is, a ratio of 4:1. They state that "all data confirms the general observation that there is more rheumatic disease in the families of rheumatic children than in the general population". They summarize their findings by saying that "they are indicative of a strong familial tendency to the disease. The fact that such a familial tendency has been manifest for at least three generations is strongly suggestive of the existence of a constitutional susceptibility to the disease. This does not eliminate the possibility that exposure is also an important factor."

Appendix VI.

Trauma as an Ætiological Factor.

In the analysis of the case records, and in patients observed during the period of this investigation it was found that by far the commonest initiating factor of the rheumatic attack was a preceding upper respiratory infection, usually an acute tonsillitis. In a few, however, the attack was apparently brought on by an injury to a joint occurring a short time before. Twelve cases illustrating this sequence were encountered (Table XI).

Swift and Cohn[21] draw attention to the possible influence of both physiological and physical trauma upon the localization of rheumatic lesions. They state:

> While rheumatic arthritis usually occurs first in the knees and ankles, it more often localizes early in the hands and arms in the case of needleworkers and laundresses.

They also mention that they have observed cases in which physical trauma to a joint was followed by rheumatic inflammation in that joint, and state that "in others a bruise apparently induced a focal relapse which was subsequently followed by migratory polyarthritis".

It is to be noted that subcutaneous nodules occur most commonly in parts subjected to physiological stress, such as tendons and their sheaths where they pass over joints, or over structures on which pressure is frequently exerted, like the olecranon or the patella. Massel, Mote and Jones[22] artificially induced subcutaneous nodules in rheumatic individuals by irritating areas previously injected with the blood of rheumatic patients. These workers have suggested the influence of trauma in the localization of lesions.

Although it is considered probable that the bases of all the valves of the heart are acutely inflamed in the early active phase of rheumatic carditis, it has been suggested that the mitral and aortic valves, especially the former, most frequently become scarred and deformed because they are the valves subjected to the greatest physiological trauma. The accompanying table is a list of cases met with in this investigation, in which trauma was apparently the initiating cause of the rheumatic attack. Care was taken to exclude any case in which the factor of a recent upper respiratory infection, such as tonsillitis, was also present.

In most of the cases, soon after trauma to the joint, an effusion of varying degree developed, and the patient was first treated for traumatic synovitis. Some hours or a few days later, symptoms of acute rheumatism occurred, several of the larger joints being successively involved in an acute inflammatory process, and in some cases carditis followed. The following hypothesis is suggested to account for the occurrence of acute rheumatism after trauma to a joint. As the result of injury a joint is likely to have its resistance to infection lowered, depending in part on the synovial effusion which commonly ensues, as this offers a suitable medium for the growth of organisms. In a person predisposed to rheumatic infection it seems possible that acute rheumatism can start in such a joint and subsequently spread to involve other joints and the heart, as commonly occurs in the disease. This is likely to happen when there is present some focus of rheumatic infection from which the virus or organism is able to migrate via the blood stream and establish itself in the injured joint. The common focus of infection in acute rheumatism is the naso-pharynx, especially the tonsils; that the infecting agent may lie dormant in these parts and be reactivated by some factor is suggested by the occasional occurrence of juvenile rheumatism following on tonsillectomy. Relapses in the disease are common, and the twelve cases quoted are of interest, for they lend evidence to support the theory that the infecting agent in juvenile rheumatism may remain resident in the body for long periods without causing symptoms, and that it may be set free from time to time to initiate an attack by various factors, of which the commonest might possibly be hæmolytic streptococcal infection.

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Table XI.

Case.	Age.	Sex.	Trauma.	Time Interval before Rheumatic Attack.	Rheumatic Manifestations or Complications.	Previous Rheumatic History.
1	5½	M.	Kick on right knee.	3 days.	Migratory polyarthritis; carditis.	Nil.
2	6	M.	Kick on right ankle.	24 hours.	Migratory polyarthritis; carditis; *erythema marginatum*.	Acute rheumatism once before.
3	25	M.	Injury to metatarso-phalangeal joint.	24 hours.	Migratory polyarthritis.	Several attacks of acute rheumatism and one of chorea.
4	16	M.	Sprained wrist.	48 hours.	Migratory polyarthritis; carditis.	Acute rheumatism seven times previously.
5	16	M.	Twisted right ankle.	4 days.	Migratory polyarthritis; rheumatic nodule; carditis.	Several attacks of acute rheumatism before.
6	13	M.	Injury to right ankle.	48 hours.	Migratory polyarthritis; carditis.	Acute rheumatism once before.
7	22	M.	Injury to right knee.	48 hours.	Migratory polyarthritis.	Nil.
8	16	M.	Injury to ankles and shoulders.	36 hours.	Migratory polyarthritis.	Nil.
9	36	F.	Hard use of arms in washing clothes.	6 hours.	Migratory polyarthritis; *erythema marignatum*.	Acute rheumatism when a child.
10	13	F.	Wrenched right wrist.	4 hours.	Migratory polyarthritis; carditis.	Nil.
11	18	M.	Left knee run over by cart.	3 days.	Migratory polyarthritis; carditis.	Nil.
12	29	M.	Injury to left knee.	48 hours.	Migratory polyarthritis.	Nil.

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Reports of Cases.

A PROCTOLOGICAL PROBLEM.

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THE rush of routine duties not infrequently leads some of us to take apparently obvious things for granted; and to those able to follow their cases to autopsy, the *post mortem* room offers from time to time a salutary corrective. The habit of making a systematic, even though brief, review of the diagnosis would lessen the need for any such correction. The following case will show that the "*P*" of the student's mnemonic "*Pint*" ("parasitic", "inflammatory", "neoplastic" and "traumatic") has not yet become of merely academic interest.

Clinical History.

The patient, J.P., aged forty-five years, was referred to the Austin Hospital from the Royal Melbourne Hospital with a diagnosis of carcinoma of the rectum. The following history is based on our own records, and the reports furnished by the Royal Melbourne Hospital (denoted where necessary by the initials "R.M.H.") on the two occasions he was referred by that hospital to us.

His history commenced about eighteen years ago with straining on defæcation accompanied by passage of blood and mucus, and, later, diarrhœa and rectal pain. He did not consult a doctor until eighteen months after the onset of symptoms, and six months later he was referred to the Royal Melbourne Hospital. Operation disclosed a fleshy growth of the rectum, "probably carcinoma" on section (R.M.H.). Deep X ray therapy was given to the lesion at the Royal Melbourne Hospital 2, 13, 14, 16 and 17 years after the first symptoms. He had been able to work for only four of the eighteen years of his illness.

Fourteen years after the onset he complained of pain in the right hip, and X ray examination disclosed "osteoporosis right ischium and lower ilium" (R.M.H.), and two and a half years later, after further X ray examination, he was referred to the Austin Hospital as incurable because of bone metastases. X ray examination at the Austin Hospital disclosed radiographically normal lungs. The report on X ray examination of the pelvis (see Figure I) states that there were extensive destructive lesions in the bones comprising the hip joint. Radiographically the appearance suggested an inflammatory rather than a neoplastic process.

The patient stated that a rectal examination was made under general anæsthesia; but unfortunately details of this are lacking.

After three months' treatment along general lines he was discharged clinically improved, but six weeks later reported at the Royal Melbourne Hospital complaining of pain in the lower part of the right leg, and much discharge *per rectum*. Wasting of the thigh and shortening of the leg were present. Some sixteen months later, after one more course of deep X ray therapy at the Royal Melbourne Hospital, he was again referred to the Austin Hospital, as stated above. At the time of his admission he had a fairly severe cough, which he said had commenced two months before, when he had got wet and "caught a chill". The cough caused considerable abdominal pain.

Examination disclosed no abnormality of heart or lungs. The liver was impalpable. There was a left inguinal colostomy. The right thigh was flexed and internally rotated, but not fixed, and it presented some muscular wasting. An attempt at rectal examination showed some spasm of the anal sphincter, but caused such severe pain that it was abandoned. In view of the two thorough examinations which had been previously made, it was assumed that this pain confirmed the previous findings.

There was no reaction to the Wassermann test. No tubercle bacilli were found in the sputum. The X ray report on the pelvis (see Figure II) was as follows:

> Metastases in right half of pelvis with destruction of acetabulum and inward dislocation of femur (arthrokatadiasis). Metastases also in upper end of femur. Slow progression of metastases since previous report.

During the first two to three weeks his cough improved considerably; but, as it did not disappear, pulmonary metastases were suspected, and a skiagram (Figure III) showed: "area of dulness toward lower half of right lung; appearances consistent with metastases and associated atelectasis and some inflammatory changes". A subsequent right lateral skiagram confirmed an area of dulness posteriorly in the right lower lobe extending from apex to base.

After five to six weeks he experienced troublesome diarrhœa from the colostomy, which no symptomatic treatment completely controlled. His sputum also was fairly profuse and purulent in appearance. Two weeks after the onset of diarrhœa he died. His temperature

whilst in hospital remained within normal limits, and his pulse rate varied from 40 to 104 beats per minute, usually 55 to 70.

Post Mortem Findings.

At autopsy the posterior and basal parts of the left lung were seen to be very œdematous, the lung being otherwise healthy. The right lower lobe was adherent to the lateral thoracic wall, and was riddled with abscesses, the largest being about three centimetres in diameter. There was no bronchial obstruction or gross evidence of malignancy. The liver was soft and engorged, but there were no carcinomatous deposits, nor were there any in other abdominal viscera, or in the para-aortic glands. There was evidence of a recent generalized fibrinous peritonitis. The intestines were otherwise normal. The rectal lumen was diminished in one place to about the size of a pencil, but otherwise the organ was normal except for a very minor degree of polyposis. Incision over the right pelvic bones disclosed, however, a mass of hydatid daughter cysts, and further dissection showed that the whole right side of the pelvis, and the right femur, were riddled with hydatids, both inside and around all of the bones and within some of the muscles.

This surprising finding led, naturally, to a review of the whole case; and through the courtesy of the Royal Melbourne Hospital we were given further information about the patient's past condition. He had been admitted to that hospital twelve years previously as he wished to have closed the colostomy which had been established five years previously. The honorary surgeon himself made the rectal examination, his finding being that:

> There was a superficial stricture which could be dilated, but above that a dense fibrous stricture which was impassable, and adherent beyond bowel. A proctoscope was inserted, and a section taken from the lower fibrous lining. The growth was felt close to bladder wall and obviously not removable.

The patient was therefore discharged. Subsequent biopsy report stated: "no evidence of carcinoma in piece of tissue sent".

It was unfortunate that, after a number of years, this report was inadvertently stated as "probably carcinoma", for that made one more item in the rather extraordinary chain of coincidences, including the apparently confirmatory lung condition, on account of which the old diagnosis was not seriously questioned, apart from our comments on the long duration of the illness.

The Austin Hospital pathological reports subsequent to autopsy were, in brief: inflammatory changes in the lungs; no evidence of malignant disease in the rectum, but chronic inflammatory changes in mucosa and submucosa.

Discussion.

The points of special interest in this case are, first, the manner in which hydatid disease of bone can mimic malignancy, and, secondly, the way in which this rectal condition maintained for so long and so convincingly the appearance of a primary carcinoma.

Hydatid disease of bone has not, perhaps, been described so often as to render out of place here a brief description of the radiological findings. Thus in Geschickter and Copeland's comprehensive 800-page treatise on bone tumours[1] echinococcus cysts do not appear to be mentioned at all, even as a differential diagnosis. The relative order of frequency with which various bones are affected is stated by Dew[2] to be humerus, femur, vertebræ, tibia and innominate; and by Brailsford[3] as pelvis, femur and vertebræ. Other bones are also occasionally involved. The combination of femur and pelvis with destruction of the acetabulum—the lesions found in our own case—was described by Kienbock and Mayer[4], quoted by Brailsford.

A hydatid cyst may occur in soft tissues and by expansion cause erosion of bone. It may occur in bone, giving rise to destruction without deformity, or rarely to the formation of a cystic expansion of the bone. In involved vertebræ collapse is rare, and extra-osseous expansion the rule. This expansion is apt to cause pressure on nerve trunks or on the spinal cord. In other cases, apart from discovery during a routine X ray examination, the features which will lead to discovery will usually be a swelling, pain from pressure on nerves, or a spontaneous fracture.

The X ray appearance is that of progressive destruction at the site of the cyst, which often has a multilocular appearance. Bony septa are often seen in the skiagram, and these may later become detached and absorbed. In Dew's experience the osseous destruction without subperiosteal thickening and total absence of "bony reaction" are important features, although Brailsford states that the bony walls may show a reactive sclerosis.

Conditions with which the disease may be confused are fibrocystic disease, myeloma and simple cysts. Should extra-osseous extension occur, cold abscess or cystic sarcoma may be simulated. Sometimes lime salts may be deposited in the hydatid cysts, and Brailsford considers that the appearance of these in the skiagram will help to differentiate from the somewhat similar osteoblastoma and hæmangio-endothelioma.

In the case reported here the cystic appearance is less clearly defined, and that of a secondary carcinoma is closely simulated.

Acknowledgement.

I wish to express my gratitude to Dr. E. T. Cato, now Squadron Leader of the Royal Air Force, for permission to publish details of this case, and to Dr. Alan Mackay for permission to reproduce the skiagrams. I should also like to thank Mr. Cecil Young for the excellent photographs of his original films.

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LARGE CALCIFYING TUMOUR OF THE THIRD VENTRICLE OF THE BRAIN.

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Tumours in the third ventricle of the brain are still not often diagnosed during life and successfully removed and calcified tumours in this situation are of some rarity, so that the present case is thought worthy of report.

A youth, aged eighteen years, employed as a messenger, noticed some six months before he was referred by Dr. Levy to the Royal North Shore Hospital that his right leg was becoming stiff and clumsy and that his right arm was slightly difficult to manage. These symptoms gradually became more obvious and he found that he could not kick a football. He stumbled frequently. He had great difficulty in writing and his handwriting became almost indecipherable. He could not shave himself nor

cut up his food. He had always been in the best of health previously and still felt very well, except for inability to concentrate on his examinations at the post office and excessive tiredness at night.

On examination, he was found to be robust and mentally quite normal. He was right-handed. Speech was normal. There was obvious right-sided facial paresis, and weakness of the right side of the tongue and in the right trapezius and sterno-mastoid muscles was noted. There was nystagmus, especially when he looked to the right, the quick component being to the right. The cranial nerve functions were otherwise normal. The fields of vision were not restricted. There was no papilloedema, but the right optic disk was slightly paler than the left. The pupils, which had been dilated with the homatropine drops kept for this purpose, remained dilated for a week. The eyesight was good. The sensibility to pain was unquestionably slightly impaired over the whole of the right side of the body. The right arm and leg were ataxic. The right arm tired rapidly, drooped when held extended and exhibited adiadochokinesia. The tendon reflexes were much exaggerated on the right side of the body and Babinski's plantar reflex sign was present on the right side. The abdominal reflexes were absent on the right side. The patient walked with a limp, swinging his right leg and dragging the toes. The systolic blood pressure was 162 and the diastolic 100 millimetres of mercury. No abnormality was detected in the head, ears, musculature, sexual development or elsewhere. The blood picture was normal.

Clearly his trouble was in the brain. The cerebro-spinal fluid was clear and with the patient lying on his side it rose in a manometer to a height of 190 millimetres; it contained one lymphocyte per cubic millimetre and 0·070 gramme of protein, 0·050 gramme of sugar and 0·738 gramme of chloride per 100 cubic centimetres. The cerebro-spinal fluid did not react to the Wassermann test; neither did the blood. The presence of a cerebral tumour would have been postulated at this stage, but an X ray examination of the head had already revealed a calcified mass larger than a walnut right in the middle of the brain.

Dr. Paul Tillett's laconic report on the skiagrams was: "Calcifying ependymoma of the third ventricle." This diagnosis was subjected to close scrutiny. Pineal tumour was considered, but the shadow was not quite in the mid-line and was slightly anterior to and above the normal position of the pineal body. Calcification in the falx was unlikely on account of the patient's youth, and the shadow was well below the level of the falx. Calcification in the thalamus has been reported, but would not transgress the mid-line. Tuberculomata are usually more peripheral in the cerebral substance. The patient and every member of his family reacted very sharply to intracutaneous injections of tuberculin, but skiagrams of their chests revealed no sign or vestige of tuberculosis.

Air encephalography was now performed by the spinal route. Little of the air entered the ventricular system, but parts of the lateral ventricles were outlined and also the anterior part of the third ventricle. Stereograms showed plainly that the tumour was in the third ventricle (Figures I and II).

The propriety of an operation to remove the tumour had now to be considered. Without such an operation no improvement could be expected; but the patient was not totally incapacitated and the risk of operation was thought to be considerable on account of the depth and size of the tumour and its difficulty of access. The surgical author thought the risks of operation less formidable to the patient than the prospects of continued possession of the tumour, and suggested operation by the posterior approach of Dandy, as the tumour was well back in the third ventricle. It was decided to advise the patient accordingly, and he was transferred to the neuro-surgical unit of the Royal Prince Alfred Hospital.

The operation was performed in two stages under anæsthesia administered by Dr. S. V. Marshall. "Avertin" (125 milligrammes per kilogram of body weight) was employed as a basal narcotic, and ether was given in a closed circuit with carbon dioxide absorption, the Magill intratracheal tube and a pharyngeal pack being used; air was employed as the vehicle, with a continuous oxygen inflow of 250 to 300 cubic centimetres per minute throughout. Cyclopropane was used for the control of generalized clonus and rigidity in the early stages and as a substitute for ether towards the end of the operation.

The patient was placed in the prone position with head flexed, and a large parieto-occipital bone flap was raised on the right side. The *dura mater* was then incised and reflected towards the mid-line. The right lateral ventricle was tapped in order to reduce the bulk of the cerebral hemisphere which had to be retracted laterally from the *falx cerebri*. Numerous veins were encountered running from the medial aspect of the cerebral hemisphere towards the superior longitudinal sinus, and these had to be clipped and coagulated before division. At last the splenium of the *corpus callosum*, the great cerebral vein running into the straight sinus and the superior surface of the *tentorium cerebelli* could be clearly seen in a dry field, and the hard tumour could be felt through the *corpus callosum*. The next step was to split the *corpus callosum* in the mid-line to gain access to the third ventricle; but as the patient had now been four hours on the operating table and vasomotor instability was evident it was decided to adjourn the operation to a second stage. It was thought that such adjournment was not unsafe in the absence of internal hydrocephalus.

The patient was none the worse after the first stage, and a fortnight later the head was reopened. The anæsthetic technique was similar. The *corpus callosum* was again exposed and divided by endothermy in the mid-line. The hard tumour was then encountered lying free in the cavity of the third ventricle, attached to the roof and surrounded by a thin, soft, hyaline-looking cortex. This was removed with the tumour, except in the habenular area and near the opening of the cerebral aqueduct, where the tumour was shelled out of this covering for fear of damage to the ependyma in this region, which might cause internal hydrocephalus. The tumour itself felt and seemed like bone; it was friable in places and some of the outlying nodular parts broke off during manipulations to deliver it. Hæmorrhage from the surrounding chorioid plexus was kept well under control. The internal cerebral veins were seen, but not damaged, and only once for a few moments was any serious venous bleeding encountered; this was controlled by "pattie" pressure. After removal of the tumour, however, a quite large clot was sucked out of the anterior portion of the cavity of the third ventricle and the interventricular foramina were then clearly seen. After hæmostasis had been secured the right cerebral hemisphere was allowed to fall back and the bone flap was replaced after careful closure of the *dura mater*. This second stage of the operation lasted five and a half hours, but was well stood by the patient with the aid of a transfusion of 600 cubic centimetres of citrated blood. The blood pressures were sustained at normal levels while the pulse became progressively slower; shortly before the end, however, a sharp fall of blood pressures and rise of pulse rate occurred, but control of this change was readily effected by an increase in the rate of the slow transfusion of blood.

Convalescence was uneventful. Lumbar puncture was performed once a few days later when some clear xanthochromic fluid under a pressure equal to 150 millimetres of water was removed. Recovery of function was rapid, and within a few days full power had returned to the right arm and leg. The patient was soon able to feed himself and the ataxia rapidly disappeared. He was able to walk on the tenth day after the operation and left hospital on the fifteenth.

The tumour after removal was seen to be somewhat lobulated and completely calcified. The main mass (after several nodules had broken off) measured 4·5 by 3·0 by 2·5 centimetres and weighed 30 grammes (Figure III).

Sections were prepared from some of the loose portions and from the soft cortex surrounding the main mass (Figure IV). These were examined by Dr. G. V. Davies,

FIGURE III.

pathologist to the Royal Prince Alfred Hospital, who reported as follows:

> The tumour has the structure of a papilloma of the choroid plexus with a very large amount of

FIGURE V.

calcification. The calcified masses are very irregular in shape and between them are villous processes of tumour growth, consisting of a fibrous core covered by columnar epithelium. The epithelium has not the appearance of malignancy. In places there is a capsule containing large blood vessels and also irregular spaces containing small nodules with concentric lamination like psammoma.

The soft layer surrounding the tumour would seem to indicate that the tumour was extra-ependymal in origin, but had become invaginated into the third ventricle.

A search of the available literature has produced a description of a similar tumour by Dandy in his book "Benign Tumours in the Third Ventricle of the Brain" (Case IX, Figure LXXXV). In this case, however, the cerebral aqueduct had apparently been blocked with resulting enlargement of the ventricular system and increase in intracranial pressure. Difficulty from hæmorrhage from communicating veins between the cerebral hemisphere and the superior longitudinal sinus was encountered, and despite a successful removal of the tumour death occurred suddenly some three hours later, presumably from hæmorrhage from this source. The photomicrograph of a section of this tumour closely resembles that submitted with the present report (Figure IV).

The appearance of the patient six weeks after the operation can be seen from Figure V, which shows the site of the bone flap. Examination of his nervous system then revealed no abnormality, and his handwriting had improved greatly (Figure VI). An X ray examination of

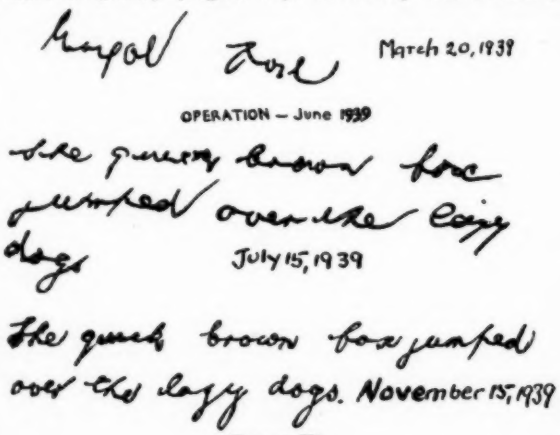

FIGURE VI.

his skull revealed a small area of residual calcification in the vicinity of the pineal gland and the bone flap in position (Figure VII). Eight months after the operation the patient's condition was still satisfactory and he had returned to work.

Reviews.

A MANUAL OF OPHTHALMOLOGY.

MAY AND WORTH'S "A Manual of Diseases of the Eye" is in its eighth edition.[1] Its growing popularity is demonstrated by the fact that it has been reedited three times and reprinted once. The latest revision has been made by Montague Hine in a manner that is in keeping with the initial aims expressed in the preface of the first edition: "to present a concise, practical and systematic manual—intended for the student and the general practitioner of medicine". It has been translated into ten foreign languages, the latest being Portuguese and Urdu. At present there is no better book for those who are commencing a study of ophthalmology. The use of "retinopathy" instead of "retinitis" is wise. One wonders how long it will be before this term is universally adopted.

[1] "A Manual of Diseases of the Eye", by C. H. May, M.D., and the late C. Worth; Eighth Edition, revised by M. L. Hine, M.D., F.R.C.S.; 1939. London: Baillière, Tindall and Cox. Demy 8vo, pp. 546, with illustrations. Price: 16s. net.

The Medical Journal of Australia

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THE SECOND AUSTRALIAN IMPERIAL FORCE AND ITS RECRUITS.

It would be difficult to under-estimate the importance to an army of an efficient medical service; the truth of this statement has in all probability never been more clearly shown than in the history of the First Australian Imperial Force during the war of 1914-1918. Efficiency in an army medical corps must depend ultimately on its possession of an administrative head, a director-general, who is well informed, who knows what he wants and who has enough punch and determination to get it. Obviously the way of a director-general may not be easy. If he is efficient he will choose as his assistants capable men who are not time servers and who are not politically minded; he will then have no reason to fear that his plans will be put into execution in any way other than that of his own choosing. But outside his own department are army headquarters on the one hand and political bosses on the other. History shows that a director-general may expect interference from both these quarters. The combatant officer naturally wants to keep his fighting units at full strength and he may not see eye to eye with a medical head who insists on a rigid standard of fitness. The politician also will show the same lack of understanding when he deals with recruiting and the reinforcement of the firing line. Thus differences of opinion arise. Though a director-general of an army medical service is responsible for the setting up of standards by which men shall or shall not be judged fit, the application of the standards and the acceptance of recruits are in the hands of the examining medical officer. The responsibility of medical practitioners who examine recruits and the need for care in examination were emphasized in this journal by Lieutenant-Colonel Eric L. Cooper and Lieutenant-Colonel C. E. Wassell in the issues of December 16, 1939, and January 6, 1940, respectively.

Recruiting for the Second Australian Imperial Force has been going on for many months. As far as can be gathered the examinations have been carefully made and proper discrimination has been shown. In a recent issue attention was drawn to the routine radiological examination to which recruits have been subjected and to its value in the detection of pulmonary tuberculosis. That the Director-General of Medical Services has been able to introduce this searching method of examination is an indication both of his far-sightedness and of his ability to impress on the Minister and on the other army authorities the need for thoroughness. There is no doubt that by this means many persons have been saved from the lighting up of a tuberculous focus, non-tuberculous soldiers have been saved from being infected by their comrades, and the Commonwealth has been saved from having to pay pensions at a later date on account of tuberculous infection. But there is doubt in the minds of many on account of what appear to be hardships inflicted on certain individuals. Some men have resigned from civilian positions on being accepted for service, they have adjusted their private affairs in the hope of being sent abroad, and they have made plans for their dependants during their absence, only to find after a lapse of many weeks that they are declared to be tuberculous as a result of a belated X ray examination. Nurses too have given up positions on being accepted for service, have spent consider-

able sums of money on uniforms and other necessary equipment, and have at the last moment been told that they could not be sent away. These people would appear to have just cause for complaint.

Routine miniature X ray examination has but recently been introduced. Lieutenant-Colonel Eric L. Cooper, who is on the staff of the Director-General of Medical Services, in addressing the Victorian Branch of the British Medical Association on November 1, 1939, said that radiological examination would almost certainly become the routine method for the permanent forces. He also referred to the development of miniature radiography, which would in the future reduce the cost of chest skiagrams. Obviously by the time this form of examination could be introduced many persons were already accepted for service, but once they were discovered to be tuberculous it was right that they should be prevented from going abroad. We cannot believe that a service which is so far-sighted as to adopt miniature radiography as a routine method of examination will not, now that facilities are available, use it in its proper place, before the recruit is accepted and allowed to dislocate himself from his civilian surroundings. This, of course, is not sufficient. Those who have already suffered hardship by rejection at the last moment, when they were to all intents and purposes accepted for service, should receive consideration. This is outside the province of the medical service; but the appropriate authority, presumably the Defence Department itself, should take action at once. Common sense and common justice demand it, to say nothing of the prospects of successful recruiting in the future.

Current Comment.

THE DIAGNOSIS OF RHEUMATISM.

AMONG those who have filled the position of senior physician to the Hospital for Sick Children, Great Ormond Street, is W. B. Cheadle. As long ago as 1888, Cheadle, in the Harveian Lectures, discussed the rheumatic state and in his subsequent publication wrote:

> I was asked by Mr. Kiallmark to see, in consultation with him, a little boy of six suffering from relapsing tonsillitis, in whom he had discovered, to his dismay, a distinct apex murmur. The child had had previous attacks, and Mr. Kiallmark had judged them to be rheumatic. The boy has had no arthritis, and there is no distinct rheumatic family history. The tonsillitis rapidly got well under salicylate of soda, recurring three times when this medicine was omitted. The murmur is a soft one, audible over the tricuspid area, not at the apex. I am doubtful whether it is organic, but I shall be much interested to learn its future course.

Despite the passage of fifty years, most physicians will still readily and ruefully admit the difficulty that Cheadle found. The frank case of rheumatic fever or of carditis is simple to place; but not so the all too frequent case of "growing pains", of recurrent tonsillitis or of vague ill health accompanied by an apical heart murmur. Is the condition really rheumatic? The bacteriologist cannot help. The evidence of the immunologist is vague and circumstantial. Electrocardiography can find changes that are at the best inconstant and indefinite. Even the hæmatologist with blood counts and sedimentation rates can find only changes common to a number of diseases. In this age of laboratory aids the diagnosis rests essentially on clinical methods, and the clinician must often echo the words of Cheadle: "I shall be most interested to learn its future course."

He can obtain some guidance, however, from two recent discussions. Much has been written of the significance of pains in the limbs in relation to childhood rheumatism, and pædiatricians have repeatedly insisted that "growing pains" are not always rheumatic. M. J. Shapiro,[1] of Minneapolis, points out that a large number of children have severe muscular pains in the extremities during childhood and adolescence, but do not exhibit the symptoms of rheumatism and do not develop heart disease. The children with non-rheumatic pains, he points out, are well nourished, healthy and frequently belong to well-to-do families. The pains are in the muscles of the legs and thighs, and rarely in the arms. They usually occur at the end of the day, especially when the child goes to bed, and may even wake him soon after he falls asleep. Next morning the pain is gone and the child has no limp. The pain is unrelated to climatic changes. There is usually no family history of rheumatism. The other symptoms of rheumatism—joint pains, fever, anæmia, failure to gain weight, nose-bleeds and skin rashes—are absent. The blood sedimentation rate and leucocyte count are normal.

Children with subacute rheumatism, however, usually first complain of pains after an upper respiratory infection or one of the contagious diseases. The pains are localized in the joints, often of arms as well as legs, are most severe when the patient gets out of bed in the morning, are worse on movement, and affect the use of the limbs. They last all day, especially if the weather is cold, and become easier when the patient becomes warm in bed. The joints may be slightly tender, swollen and warm to touch. The children, often of the

[1] *Journal of Pediatrics*, March, 1939.

poorer classes, are frequently of rheumatic families. Pallor, poor nutrition, slight fever, mild leucocytosis and increase in blood sedimentation rate are common. Evidence of carditis may or may not be found. The pains occur in recurrent attacks, each lasting several days. "We have then", writes Shapiro, "on the one hand a group of healthy children who complain of leg pains, especially at night, while on the other hand we are dealing with sick children who have all the symptoms of a chronic infectious disease." Confusing borderline cases, he admits, do exist, but in the main the distinction is clear.

The diagnostic significance of a precordial murmur is discussed by M. M. Maliner and Irving Okin.[1] Every loud murmur they regard as indicative of organic heart disease. In the class of patient under consideration, these will be due either to rheumatic or to congenital disease. The faint murmur, no matter what its precise location, may be insufficient ground for a diagnosis of an organic lesion, and many clinicians are content to repeat the words of Cheadle. Maliner and Okin, however, have devised a method of accentuating heart murmurs which has helped them to sort out these difficult cases. The child with a faint precordial murmur is placed in a recumbent position, the heart sounds and murmurs are noted, and the pulse rate and blood pressure are recorded. Epinephrine, 1 in 1,000, is injected into the deltoid region. The child under eight years receives six minims, and over eight years eight minims. Immediately and every five minutes thereafter for half an hour the same auscultatory and instrumental findings are recorded. Changes in intensity, quality, location and duration of the murmur are noted. If the murmur remains unchanged or decreases in intensity it is regarded as functional. A systolic apical murmur which increases in intensity and becomes transmitted toward the axilla is regarded as indicating mitral incompetence. If a diastolic apical murmur appears as well, some degree of mitral stenosis is presumed. A murmur which becomes accentuated and is localized along the left sternal border suggests a patent interventricular septum. The test also unmasked a case presumed to be one of partial coarctation of the aorta. One might expect it to reveal the occasional mitral presystolic murmur or the aortic to-and-fro murmur that is missed by routine methods of examination of children.

Five years ago this test was applied to a group of 32 children whose precordial murmur was regarded as functional. As a result, 23 were regarded as suffering from organic heart disease and in nine instances the diagnosis remained functional. Of the 23 children, 11 now have developed definite organic heart disease, three have defaulted from the clinic, and in nine no changes have taken place. Of the nine regarded as a result of the test five years ago as having functional murmurs, five are still looked upon as having functional murmurs, three have been discharged from the clinic as free of heart disease, and one has defaulted. Thus in the relatively short period of five years the gravity of the murmur suggested by the test has proved justified in 47·8% of the children, and no condition dismissed as functional has proved organic. The results have been most accurate in older children. Of a group of 37 patients recently tested, 14 have been classified as suffering from functional and 23 as suffering from organic murmurs. The use of amyl nitrite for a similar test has been found quite unreliable by the authors.

Until research into the rheumatic state provides a diagnostic method, the results of which will allow no doubt, the clinician's art remains his arbiter. The discussions of Shapiro, Maliner and Okin make useful contributions to that art.

[1] *Journal of Pediatrics*, January, 1937.

A POISON FOR SILVERFISH.

Most people's knowledge of silverfish is confined to their depredations among books and photographs in the home. Those who wish to know something about these curious pests will find an informative article in *The Australian Museum Magazine* of June 30, 1938, written by Keith C. McKeown. The silverfish that infest our homes are immigrants from other lands; the native Australian insects remain in the bush. The most familiar house-frequenting forms are the European *Lepisma saccharina*, which has a rich silvery lustre, and the African *Stenolepisma longicaudata*, which has a whitish cream colour and has not the metallic appearance of *Lepisma saccharina*. Silverfish do not pass through the egg, larva, pupa and imago stages. There is an egg, but when the egg is hatched the baby insect is in all essentials a silverfish. As the insect grows it sheds its coat several times, and after the last shedding it is a sexually mature adult. It takes several years for the insect to develop from egg to adult. Silverfish are nocturnal, and by means of strong toothed mandibles they devour bookcovers, pictures, photographs and wallpapers, being attracted chiefly by the starchy dressing. In the *Health Bulletin* of the Commission of Public Health, Victoria, Numbers 59 and 60, 1939, we read that the Council for Scientific and Industrial Research has released the formula for an effective poison, which is more attractive than the natural foods. One ounce of flour is mixed with one and a half ounces of sugar and ten ounces of warm water into a paste, and into this one and a quarter ounces of barium fluosilicate is stirred while the paste is warm. The paste is spread onto both sides of large sheets of thin cardboard, which is cut into pieces two and three inches square. A fly spray (kerosene pyrethrum) is well used in infested parts of the dwelling. Ten to twenty baits are set out near the lurking places of the silverfish—but out of the reach of children, for barium fluosilicate is a poison. The baits should be left for some weeks and the process repeated if reinfestation occurs.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

Tanning from Ultra-Violet Irradiation Brought Out by Blood Transfusion.

E. O. Jodar (*American Journal of Diseases of Children*, November, 1939) reports the case of an infant, aged nine months, which was anæmic and still very pale after eighteen treatments with a mercury vapour lamp at weekly intervals. A blood transfusion was given, and next morning it was noticed that the skin had a light tan colour with a pale area around each eye corresponding exactly with the goggles worn during the irradiation. It is suggested that the lack of the precursors of pigment (tyrosine and phenylalanine) in the blood in quantities sufficient to allow for the usual tanning by ultra-violet irradiation is entirely compatible with life. It is also suggested that the cells of the skin can be sensitized by ultra-violet irradiation and that the pigment-producing enzymes can be activated and can remain present, so that when the necessary chromogens are supplied by the blood of a normal person the process of tanning occurs quickly.

Metallic Elements and Blood Formation.

M. O. Schultze (*Physiological Reviews*, January, 1940) reviews recent work on the importance of metallic elements in blood formation, restricting the term "blood formation" to the formation of red blood cells and hæmoglobin. For the purpose of discussion, hæmoglobin is considered as consisting of globin, protoporphyrin IX and iron. Studies on the histogenesis of red cells indicate that there is a gradual accumulation of hæmoglobin during the development of the cell. The seat of hæmoglobin synthesis, at least in its final stages, is apparently in the nucleus of the developing cell. The author stresses the close relationship between the formation of red cells and of hæmoglobin, and states that the chronic hæmorrhagic anæmia of dogs and the nutritional anæmia of rats and other species provide the best material for the study of blood formation. Recent improvements in the field of bone marrow culture open a new approach to the study of the function of metals in blood formation. The function of iron as a constituent of hæmoglobin is, of course, evident. Since the formation of hæmoglobin and that of erythrocytes are interdependent, iron also affects erythropoiesis. The "availability" of iron compounds is discussed. The utilization of the administered iron in terms of hæmoglobin formation is generally quite low, and not directly proportional to the dose of iron. In contrast, the author states that iron injected intravenously or parenterally has been recovered almost completely as hæmoglobin iron. Another interesting fact is that the iron of orally administered hæmoglobin or hæmatin is apparently not utilized by anæmic animals. It is stated that iron absorbed in the intestine, presumably as ferrous iron, passes directly into the blood stream. The erythrocytes are apparently impermeable to iron added *in vitro*. If this is true *in vivo*, it would indicate that absorbed iron is incorporated into the maturing blood cells of the bone marrow very rapidly, because Hahn and others have shown that radioactive iron appears in the red blood cells a few hours after it has been fed to anæmic dogs. The author discusses the relationship of copper to blood formation. Copper is not a constituent of the hæmoglobin molecule. It has, however, been demonstrated that copper is essential for blood formation in animals. The results of clinical trials in man have not been uniform, as might be expected from the wide distribution of this element in Nature. The amounts of copper effective in the cure of anæmia are exceedingly small; five microgrammes of copper per day, added to a diet of milk and pure iron, suffice for rats to initiate and to maintain a steady formation of hæmoglobin and of erythrocytes. The available evidence suggests that copper is of more importance in young animals than in the adult, and that it exerts its influence in those stages of erythrogenesis preceding the reticulocyte. The relationship of cobalt to blood formation is discussed briefly, and the effects of calcium, phosphorus, germanium, manganese, zinc and vanadium are mentioned. The author states that there is at present no evidence that metallic elements other than iron, copper and cobalt have any direct effect on the formation of erythrocytes and hæmoglobin.

Functions of the Carotid and Aortic Bodies.

Carl F. Schmidt and Julius H. Comroe (*Physiological Reviews*, January, 1940) review the literature on the functions of the carotid and aortic bodies. The anatomy of these bodies is described in detail. Their physiological properties are described in the order of the historical development of the subject. As regards the part played by chemoreceptor activity in the control of respiration, two distinct viewpoints are discussed. According to one, the chemoreflex system plays a significant part in the control of breathing under all conditions. According to the other, the reflex system is too insensitive to respond to the slight changes in carbon dioxide tension or pH value of the blood associated with respiratory control under ordinary conditions; but by virtue of its great resistance to depressant agencies it comes into play as an accessory supporting mechanism in emergencies such as anoxia and unusually great increases in carbon dioxide tension. In the opinion of the authors, the truth lies somewhere between these extremes. The vasomotor and cardioinhibitory centres are affected in the same direction as the respiratory by changes in chemoreceptor activity. The author quotes the experiments of Bernthal in support of his view that the sensitivity of the chemoreflex mechanism acting on the vasomotor centre is equal to that pertaining to respiration.

Diameter of Red Blood Cells in Healthy Young Women.

E. G. Donelson, Jane M. Leichsenring and Lucille M. Wall (*The American Journal of Physiology*, January, 1940) have made red cell diameter measurements on 250 blood samples taken from 80 healthy college women, ranging in age from seventeen to twenty-four years. All blood samples were collected between 8 a.m. and 9 a.m., with the subjects in basal condition. Samples were collected from the finger tip and diluted with Hayem's solution, the counting chamber was charged, and one diameter of each of 200 round cells was measured immediately. The mean diameter was 7·35μ, the range of means from 6·53μ to 7·76μ. The mean difference between the smallest and largest cells in each of these blood samples was 3·56μ. Investigation of the frequency distribution, based on the measurement of 20,000 cells, revealed a maximum spread of 3·96μ to 10·80μ, with a mean and standard deviation of 7·31μ and 0·62μ respectively.

The Glycotropic (Anti-Insulin) Substance of the Anterior Pituitary Gland.

H. Jensen and J. F. Grattan (*The American Journal of Physiology*, January, 1940) have investigated the insulin resistance of mice after the injection of various hormone preparations. They have found that adrenotropic anterior pituitary fractions, as well as adrenal-cortical extracts and crystalline corticosterone acetate, produce a definite "glycotropic" effect. On the other hand, administration of purified thyreotropic preparations (free from adrenotropic hormone), prolactin, and "F.S.H." preparations, as well as crystalline thyroxine, fails to render the animal resistant to insulin under the experimental conditions employed. The results suggest that the glycotropic effect of the anterior pituitary is produced by the adrenotropic factor.

BIOLOGICAL CHEMISTRY.

Vitamin B_1 Deficiency in Man.

R. Goodhart and H. M. Sinclair (*The Journal of Biological Chemistry*, January, 1940) have presented evi-

dence to show that with few exceptions the amount of cocarboxylase in blood, determined by a chemical method, varies directly with the amount of total vitamin B_1, determined by a biological method. The cocarboxylase content also varies directly with the saturation of the tissues with the vitamin. In most clinical cases the determination of the amount of cocarboxylase in the blood provides a rapid and reliable method of estimating the degree of saturation of the tissues with vitamin B_1. The estimation of cocarboxylase in the blood of patients with a variety of diseases has indicated an ætiological relationship between deficiency of vitamin B_1 and "alcoholic" neuritis, nutritional neuritis, and some cardiovascular disturbances, and a possible relation of vitamin B_1 deficiency to diabetic neuritis. Deficiency of vitamin B_1 has also been found associated with cases of anæmia, subacute combined degeneration and certain psychiatric disorders. The investigations showed that definite deficiency of vitamin B_1 was not uncommon among hospital patients in England, and the authors emphasize the fact that vitamin deficiencies in man are almost always multiple and are therefore as a rule inadequately treated by administration of a single pure vitamin.

Methæmoglobinæmia following Sulphanilamide Therapy.

Several recent papers support the view that the cyanosis in patients treated with sulphanilamide is due to the formation of methæmoglobin. Rarely sulphæmoglobin is found. Irwin Vigness, C. J. Watson and W. W. Spink (*Journal of Clinical Investigation*, January, 1940), using a spectrophotometric method, found that the presence of methæmoglobin (and rarely sulphæmoglobin) explained the appearance of cyanosis. No evidence was obtained of the presence of other abnormal pigments in quantities large enough to contribute appreciably to the cyanosis. Intravenous injection of methylene blue abolished the cyanosis and the spectral distribution curve of the blood became normal.

C. L. Fox, junior, and J. E. Cline (*ibidem*) also showed the occurrence of methæmoglobin and sulphæmoglobin in the blood of patients treated with sulphanilamide. It was found that *in vitro* the conversion of hæmoglobin to methæmoglobin was produced by certain oxidation products of sulphanilamide, and evidence was adduced that this reaction also occurred *in vivo*.

W. B. Wendel, N. M. Wendel and W. W. Cox (*The Journal of Biological Chemistry*, November, 1939) performed spectrophotometric and gasometric analysis on samples of blood from nine patients with cyanosis due to sulphanilamide therapy. The results indicated that the abnormal colour was for the most part due to methæmoglobin. It was found that coloured substances present in irradiated dilute solutions of sulphanilamide rapidly converted hæmoglobin to methæmoglobin; but no evidence was obtained of the presence of these substances in the blood of patients.

Vitamin A Deficiency in Diabetes Mellitus.

J. G. Brazer and A. C. Curtis (*Archives of Internal Medicine*, January, 1940) have studied, by means of a biophotometer, the light adaptation of twenty normal subjects and twenty patients with *diabetes mellitus*, but with normal fundi. The control group showed values comparable with the normal values established by other workers. All the diabetic patients were found to have poor light adaptation, despite the fact that the blood carotene content was consistently above normal. Three patients had a history of night blindness, and nine showed clinical evidence of mild vitamin *A* deficiency by cutaneous changes. Large daily doses of carotene failed to improve the light adaptation of the diabetic patients. Comparable amounts of vitamin *A* in the form of concentrated fish liver oils, given over a similar period of time, resulted in a return of their light adaptation to normal or nearly normal within three to twenty-one days. Removal of this vitamin *A* supplement resulted in immediate relapse to subnormal values. The evidence strongly suggests that patients suffering from *diabetes mellitus* are unable to convert carotene to vitamin *A*.

Biochemical Behaviour of Lead.

A. E. Sobel, H. Yuska, D. D. Peters and B. Kramer (*The Journal of Biological Chemistry*, January, 1940) have conducted experiments designed to study the influence of dietary calcium, phosphorus and vitamin *D* on the biochemical behaviour of lead in young rats. The experimental diets varied in content of these three factors, while the lead content was held constant. All three factors were found of importance in the behaviour of lead. The results showed that the deposition of lead in the bones could be explained by postulating that lead, phosphorus and vitamin *D* formed a system of lead deposition analogous to the system of calcification. The addition of calcium tended to remove phosphorus from the system for lead deposition. It was demonstrated that simultaneous deposition of lead and removal of calcium from the bones is possible, and that increased calcification does not necessarily mean increased lead deposition. The conclusion was reached that lead deposition is directed by a system of its own, which is governed by the same laws as is calcium deposition, but does not necessarily go in the same direction. The effect of calcium on lead deposition was essentially competitive, because it tended to remove phosphorus available for lead deposition. The authors clearly demonstrated the inadequacy of the generally accepted statement that "the direction of the lead stream is similar to that of the calcium stream—that when calcium is being deposited in the bones circulating lead is also deposited in the bones, and when calcium is being pulled from the bones some lead is also liberated".

Vitamin A Deficiency.

L. Bradley Pett (*The Journal of Laboratory and Clinical Medicine*, November, 1939) has described a rapid visual test for vitamin *A* deficiency in man, depending on the recovery time after the subject has been looking at a bright light. Each testing requires less than one minute, and a patient can be tested three times within five minutes. The relation of the test to vitamin *A* has been proved by a report of experimentally induced vitamin *A* deficiency in man; by recovery on treatment of 200 patients classed as deficient by the test and by prolonged recovery times in patients with liver disease or on special diets. The effects on the result of the test of sex, age, colour of eyes, myopia, hypermetropia, astigmatism, wearing of spectacles and fatigue were found to be negligible. Of 1,600 people examined, 52% were found to be deficient in vitamin *A*, the highest incidence being found in a group of unemployed single men on relief. In general, adults were found to be more deficient in vitamin *A* during winter than they were in summer months. Attention was drawn to the possibility of vitamin *A* deficiency in special diets, particularly the Sippy or other diets used in the treatment of gastric ulcers.

Anæmia and Latent Rickets in School Children.

N. Crowley and S. Taylor (*Archives of Disease in Childhood*, December, 1939) found in school children a mild iron-resistant anæmia associated with latent low-phosphorus rickets. Twelve children, aged between six and twelve and a half years, were studied. The anæmia was either hypochromic or orthochromic and failed to respond to iron therapy. The inorganic blood phosphorus content was low, although there were no definite clinical signs of rickets. Vitamin *D*, given in two single doses of 200,000 international units at fortnightly intervals, restored the blood phosphorus values to normal in one month, but had no effect on the hæmoglobin levels. The anæmia responded to treatment with vitamin *D* and ferrous sulphate, indicating that vitamin *D* was essential in these children for the proper utilization of iron for blood formation.

British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch of the British Medical Association was held at Saint Vincent's Hospital, Sydney, on October 20, 1939. The meeting took the form of a number of clinical demonstrations by members of the honorary staff of the hospital. Part of the report of this meeting appeared in the issue of March 16, 1940.

Mammaplasty.

Dr. V. Kinsella showed two patients illustrating the operation of mammaplasty. The first patient was a mannequin and her breasts had interfered with her work. They were large, pendulous and obviously asymmetrical. In order to make the nipples symmetrical Dr. Kinsella had had to leave the operation scar on the left breast higher than the preferable submammary position. The left breast was a little larger than the right, and a second minor operation for the excision of an elliptical piece of tissue was to be performed at the patient's request, to procure symmetry. The patient was pleased with the result of the first operation and had reported the disappearance of an inferiority complex.

The second patient, a married woman, had suffered from intractable pain in the outer half of the right breast; the pain was due to chronic mastitis, and was uninfluenced by conservative treatment and physical therapy. The affected part of the breast was excised and mammaplasty was performed at the same time. The patient was pleased with the result, in spite of a post-operative wound infection (possibly due to the mastitis) which had spoiled the appearance of the skin incision.

Cystic Hygroma.

Dr. Kinsella also showed an infant suffering from bilateral cystic hygroma of the neck. Dr. Kinsella said that the condition was usually unilateral; bilateral cases were rare.

Discussing ætiology, Dr. Kinsella said that the lymphatic system developed by budding from the veins at the root of the neck. It was thought that some of these buds failed to make communication with the nervous system and were thus deprived of an outlet and of some growth-restraining influence.

Dr. Kinsella then referred to the pathology. He said that the tumours were multilocular cysts, filled with a fluid that might coagulate on exposure to the air. The skin was freely movable over them, except in the case of some children, usually under-nourished, in whom the skin was damaged by pressure. Deep processes often passed into inaccessible situations, between deep muscles, vessels and nerve trunks and into the mediastinum. If these were left behind at operation the swelling recurred.

Discussing treatment, Dr. Kinsella said that many of the swellings disappeared suddenly in the first few years of life. The larger the swelling, the greater was the possibility of its sudden disappearence. A waiting policy should be adopted. Operation was contraindicated for very young children, and physical therapy was also contraindicated (de Monchaux).

Gastroscopy.

Dr. James E. Sherwood demonstrated the Wolf-Schindler flexible gastroscope, the rubber sheath of Rogers, which permitted a localized ballooning of the sheath and consequent removal of mucosal obstructions to vision in the stomach, and Rogers's method by which a probe could be passed through the rubber sheath to enable any particular portion of the stomach to be probed under vision with the gastroscope. Dr. Sherwood then showed a patient who had suffered from duodenal obstruction and had recovered with purely medical treatment; the lesion had been observed gastroscopically as well as radiologically. Dr. Sherwood said that a condition of duodenal ileus had been considered as a possible cause, although a final diagnosis of duodenal obstruction due to adhesions was eventually arrived at.

The patient, a male, aged forty-five years, had complained of vomiting after food of two weeks' duration, with epigastric pain and loss of weight for the same period. He had undergone an operation for ruptured duodenal ulcer in 1934. A provisional clinical diagnosis of pyloric obstruction was made. On June 26, 1939, the radiologist reported that the emptying time of the stomach was considerably delayed, the stomach containing at least half the meal as a three-hour residue.

A gastroscopic examination was made on July 4, 1939. Eight ounces of bile-stained gastric juice were evacuated as a preliminary. The pyloric orifice was patent throughout the examination; it was quite fixed and did not alter in size. It had the appearance of a porthole. Very large folds, not obliterated by air inflation, were present in the body of the stomach. A provisional diagnosis of duodenal obstruction was made.

After a short period of rest and medical treatment the patient's symptoms completely disappeared. The radiologist had recently reported the presence of a deformity of the duodenal bulb, due to an ulcer close to the pylorus. The stomach was emptying satisfactorily. A further gastroscopic examination was made on August 8, 1939. Very little residual gastric juice was evacuated. The pylorus was now seen to be contracting and relaxing quite normally. The large folds were still visible in the body mucosa.

Intestinal Intubation.

Dr. Sherwood then demonstrated the Abbott-Miller method of intestinal intubation, with the object of encouraging the use of the Abbott-Miller tube. He said that it was his opinion that no hospital should be without this tube, which was simple in use and valuable in results.

The purpose and technique of introduction of the tube were most easily extracted from Pilling's leaflet on the subject. In this leaflet Pilling described the purposes of the tube as being three in number. They were: (i) the treatment and diagnosis of obstructive lesions of the small intestines; (ii) the control of the patient with intestinal obstruction, by enabling him to eat while the residue was being drawn off above the point of blockage; (iii) for use after gastro-enterostomy.

With regard to technique, Dr. Sherwood again quoted from Pilling's leaflet. He said that to intubate the small intestine satisfacforily, whether the gut showed evidence of normal function, of a mechanical obstruction or of a paralytic ileus, three factors were necessary: (i) a large tube for aspirating the intestinal contents, (ii) a thin rubber balloon at the end of the tube, (iii) a small tube for inflating the balloon. Two separate tubes were satisfactory and were often used; but the most convenient apparatus now available was a double-lumen tube having an eccentric partition throughout the length, giving one large lumen for fluid and one small lumen for air.

The air should be withdrawn from the balloon, the tube lubricated with a non-oily substance, and the tip passed through the patient's nose. As the first mark (45 centimetres) disappeared, the tip entered the stomach, and suction, which was maintained constantly thereafter, was begun. The patient should be propped up on his right side and the tube should be advanced inch by inch, as in the passage of a duodenal tube, until the third mark (75 centimetres) reached the nose. When this position was reached, a smoothly working glass syringe should be connected to the lumen leading to the balloon and ten cubic centimetres of air should be injected. If a sense of rhythmically recurring resistance was felt, the air should be left in the balloon and there should be a delay of twenty minutes. If the rhythmic resistance could still be felt, the volume of air in the balloon should be increased to 25 cubic centimetres, that lumen should be clamped, and the patient should be allowed to swallow six inches of tube every half-hour. If upon the application of the syringe no resistance was felt, there should be a delay for

half an hour, when a further attempt should be made. When no sense of duodenal contraction could be felt in a reasonable time, fluoroscopic guidance of the tube should be used.

Dr. Sherwood said that it was obvious that in many cases radiological help would be difficult to secure; fortunately, however, such assistance could be dispensed with, at least in an emergency. A simplified procedure for the introduction of a tube into the duodenum had also been described by Margenstern in *The Journal of the American Medical Association* of July 18, 1931. The tube was passed into the stomach with the patient in the sitting position. When the tube was in the middle region of the stomach, seen radiologically, the operator pressed his left hand against the patient's abdomen in the region of the greater curvature, in an upward position, to raise the greater curvature to the level of the pyloric sphincter. The tube was slowly swallowed by the patient while it was being guided towards the pyloric canal by the operator's right hand. The tube was gradually massaged into the first part of the duodenum and introduced approximately one to two inches past the spinal column. It was held in this position while the patient swallowed more of the tube, permitting a loop to form. At that time the patient's left hand was substituted for the operator's left hand, so that the greater curvature was kept on a level with the pylorus. The patient was made to lie on the right side and aspiration was begun. The loop that was formed aided in keeping the tube in place and permitted it to pass farther into the duodenum. In routine investigation work this method would prove a great time-saving factor, but naturally would appear impracticable in such an emergency as an acute obstruction.

Dr. Sherwood explained that his demonstration was given to present (i) the tube and (ii) X ray films showing the tip of the tube and balloon in the jejunum; in one case the balloon was inflated with air, and in the other case it was filled with an 8% solution of sodium iodide. His case reports so far were not numerous. In one normal case Margenstern's manœuvres were repeated and in another normal case intubation was carried out. The tube was swallowed, and one hour later bile drained freely; three hours later the inflated balloon passed into the jejunum and descended fairly rapidly. In a case of intestinal obstruction the same times approximately were worked out and the results proved eminently satisfactory. This patient, a man, who had been referred by Dr. Perry, had a ruptured transverse colon, discovered fourteen days previously. He then showed symptoms of obstruction: constant pain, distension and effortless vomiting *et cetera*. With the escape of flatus and fluid (intestinal) from the intubation tube his symptoms and signs of obstruction rapidly disappeared. In twenty-four hours his condition could be considered quite normal once more. In a further case of peritonitis it was found that though the abdominal distension and rigidity were reduced, the peritonitic process continued without response to the treatment.

Biliary Duodenal Fistula.

Dr. Sherwood then showed a female patient, aged thirty-six years, who had complained of epigastric pain off and on for two years; the pain was intermittent in character, was relieved by soda, but was not related to food. "Fullness and wind" occurred after food and nausea was frequent. There had been no history of jaundice. Stews disagreed with her.

Physical examination revealed only slight abnormality, apart from occasional tenderness to the right of the mid-epigastric line, midway between the xiphisternum and umbilicus. The results of fractional test meal investigations were well within normal limits. X ray examinations gave the following information. On March 2, 1938, no organic lesion of the stomach was seen. There was a large diverticulum in the second part of the duodenum. The radiologist suggested Graham's test, to exclude the possibility of any connexion with the gall-bladder. On March 29, 1938, the gall-bladder was not visualized on repeated examination by means of Graham's test; all the dye was seen in the large bowel. There were two shadows in the region of the fundus of the gall-bladder, which the radiologist considered would prove to be due to calculi in a non-functioning and diseased gall-bladder. A third radiological examination on August 2, 1939, revealed a large diverticulum in the second portion of the duodenum, but no organic lesion in the stomach. On September 6, 1939, Graham's test was repeated. The gall-bladder was seen to be not functioning. There was a considerable amount of barium in the region of the gall-bladder, which had remained since the last ingested barium meal on August 2, 1939, and the radiologist considered that there was probably a fistula between the gall-bladder and the duodenal bulb.

Eventration of the Diaphragm.

Dr. Sherwood's next patient was a female, aged fifty-eight years, who complained of vomiting, present intermittently for eight or nine years. The vomiting occurred about half an hour after food, and the vomitus consisted of undigested food. There was no history of dysphagia.

The patient had had typhoid fever and had also formerly suffered from "bilious attacks", consisting of headache and vomiting.

An X ray examination was made on December 14, 1938. The barium bolus was delayed in its downward passage at the cardiac end of the œsophagus, where there was a stenosing deformity and some dilatation above it. The appearances were more suggestive of stricture than of carcinoma. The radiologist suggested further examination by œsophagoscopy. On April 4, 1939, an œsophagoscopic examination revealed no œsophageal abnormality.

Dr. Sherwood said that Dr. Anderson Stuart, in discussing this case, considered that the X ray appearances were those of a congenitally short œsophagus with eventration of the diaphragm. The interesting features seemed to be the intermittency of symptoms and, above all, the invariable relief—in fact, the total disappearance of symptoms for weeks at a time—obtained by the passage of a mercury bougie. It was likely that Dr. Anderson Stuart's diagnosis of eventration of the diaphragm was the explanation of the condition.

Dercum's Disease.

Dr. Sherwood then showed a female patient, aged thirty-two years, who had complained of "pain in both sides of the face and back for nine years". She had had bilateral operations for trigeminal neuralgia, and also five alcohol injections for the same condition. In addition, appendicectomy, salpingectomy, and operations for adhesions and the removal of tonsils and adenoids had been performed.

On examination the patient's cerebration seemed somewhat retarded; an interstitial keratitis of the right eye and a left facial paralysis were noticeable. Very tender, lobulated, lipomatous masses, symmetrical in distribution, were seen on the outer and proximal aspects of both thighs. Dryness of the hair and skin and somewhat tapering fingers seemed to suggest a polyglandular disorder. The blood failed to react to the Wassermann test. Dr. Sherwood said that the tender and symmetrical lipomatous deposits, the psychogenic diathesis *et cetera*, seemed to suggest a diagnosis of Dercum's disease or *adiposis dolorosa*.

Bilateral Cervical Ribs.

Dr. Sherwood finally showed a female patient, aged thirty-five years, who complained that, three years earlier, pain, paroxysmal in character, commenced in the upper portion of the right arm; for the last twelve months the pain had been constant. The patient had had pain also in the right wrist. On one occasion she had had slight pain in her left arm. She had had numbness in her hand during the last twelve months, but no pain during the last two weeks.

On examination no wasting of the hands, forearms or arms was found; there was no loss of power. The reflexes were active and there was no gross sensory disturbance. Bilateral cervical ribs were palpable in the supraclavicular regions. The cervical ribs were further revealed by X ray examination.

A Case for Diagnosis.

DR. BRUCE HALL showed a female patient, aged fourteen years, who had recently attended at the hospital on account of fits. He said that her history and the clinical findings were interesting and called for comment. She was born at full term, the first child in the family. She sat up at the age of six months and walked at between twelve and fifteen months. Her primary dentition occurred at the normal time; but the teeth, as they came through, were very poor in quality, and later, at the age of three years, all her teeth had to be removed excepting the lower central incisors. The second dentition occurred at the usual ages; but two teeth had failed to erupt. She endeavoured to talk at the usual age, and appeared to have progressed up to a point at about the age of three years. From then on it was noticed that her speech was not developing normally, and as she became older the defect became more obvious. It appeared that she simply ceased to develop in this respect. Her vocabulary seemed to have been the chief trouble; she had not been able to add appreciably to it, but in addition she probably had some difficulty in the actual execution of speech. From then on, and indeed up to the time of the meeting, she had made conscientious efforts to learn; but she could not read words of more than four or five letters, and her spelling was bad. She went to school and did such things as drawing, needlework *et cetera* with some success, but was not so good in other subjects. The average age of the girls in her class was between ten and eleven years. In her home she was good at housework, careful and scrupulously clean. She could and did play games, but did not seem very interested in them. Menstruation first appeared at the age of ten and a half years, and the secondary sexual characteristics developed rapidly at that time. She had menstruated regularly and normally since.

Examination revealed her to be of short stature, with a noticeably small head, the circumference of which was three inches under the average for her age. Her body was that of a matured young woman, with well-developed breasts and a full and normal distribution of hair. There was an absence of enamel on some of her teeth. There were no other findings of note on clinical examination. Her vision was $^6/_6$ in each eye; the fundi were normal and the visual fields were normal. Her skeletal age was reported by the radiographers to be twenty years; the *sella turcica* was normal. A glucose tolerance test resulted in the following figures: fasting, 0·085; half an hour after receiving 50 grammes of glucose, 0·130; one hour after, 0·115; two hours after, 0·100.

Dr. Hall remarked that the girl presented a curious combination of microcephaly with mental deficiency, and skeletal and physical development advanced beyond her actual age; in other words, she had a mental age of under ten years, her actual age was fourteen years, and her physical age was about twenty years. He had not formed a definite opinion as to what was the nature of this disturbance.

Congenital Athetosis.

Dr. Hall next showed a married woman, aged twenty-four years, who had come to the hospital in February, 1939, complaining of various symptoms not relevant to the present discussion. Abnormal movements of the arms and an ataxia of speech were noted. She stated that these disturbances had been present all her life. There was no family history of similar or allied troubles. There was a history of instrumental delivery at birth and of trauma to the head at that time. She never crawled as a baby and did not learn to walk till the age of three years. She showed no real evidence of mental defect. She had had no schooling in the usual sense, but had had three years' tuition in a convent and acquired sufficient education for her practical needs. She was able to feed herself without difficulty, except when excited (she might then be "clumsy"); and she dressed herself slowly, but otherwise normally. She had married, had borne a child two years previously and managed her house and domestic affairs efficiently.

On examination persistent grimacing movements were present in the face, and athetotic movements were frequently seen involving the neck; similar writhing movements were often present in the arms, and always when the patient was under stress. Sometimes the arm movements ceased for a short time when she was unexcited and at rest. Under excitement movements of a similar nature, but less pronounced, were evident in the lower limbs.

She walked on a moderately wide base, with her left foot tending to swing in. Her gait was stiff and shaky; her knees were never fully extended in walking. Her body was not flexed at the hip joints. She limped slightly on the left side. Her right arm was slightly flexed at the elbow joint; her left arm swung free. There was nothing particularly noticeable in the wrist joints. Her head and neck moved in the choreiform manner; this was evident not only when she was walking, but at other times. Her speech was clumsy and spastic, and was further modified by the facial movements; she spoke slowly, but was easily understood.

Examination of the ocular motor nerves revealed that the ocular movements were full and the visual axes were parallel in all directions. There was no ataxia of ocular movement and no nystagmus. The pupils were equal, central and circular, and reacted briskly to light; but the left eye did not converge well, nor did the pupils constrict appreciably to accommodation. The fundi were clear. Except in so far as she had a disturbance of speech, there was no evidence of disturbance of the other cranial nerves.

Examination of the motor functions revealed that muscular power was good everywhere, and was symmetrically equal. Muscle tone might be slightly increased in the upper limbs, and was definitely increased in the lower limbs. When the patient carried out the "finger-nose test", there was some shakiness as the nose was approached; this was evident on both sides. A similar intention tremor was evident on both sides, when the "heel-knee-shin" test was attempted. The deep reflexes in the arms were all present and brisk, though on account of the athetotic movements it took a little time to elicit them. The patient gave a positive response to the finger flick test on both sides. The knee jerks were remarkably brisk, as were also the ankle jerks. The right plantar reflex was extensor in type, the left plantar reflex was uncertain. A few clonic twitchings could be felt when the patella was depressed on both sides, and at both ankle joints; but definite clonus was not obtained. The abdominal reflexes were present. There was no evidence of any sensory disturbance whatever.

Dr. Hall remarked that although there was a history of birth injury in this case, the disturbance was remarkably symmetrical in its distribution, and was therefore more likely to be classified under the cerebral diplegias. Congenital athetosis with spastic diplegia seemed an adequate diagnostic description of the condition.

Cerebral and Brachial Plexus Lesions.

Dr. Hall's third patient was an unemployed man, aged twenty-one years. One year and nine months earlier he had been involved in a motor car accident. He was admitted to Prince Henry Hospital and remained unconscious for six weeks. During that time he had an operation for depressed fracture of the skull. On regaining consciousness he found his left arm in an aeroplane splint and his left leg in a long splint. He remained in hospital for three months in all, and both splints were removed at the time of his discharge. He noticed that his arm seemed weak, both before and after removal of the splint, and he had no movement in the fingers. The left leg appeared to him to be unaffected. He was normally left handed. During his recovery he attempted to educate himself to use his right hand, and this was associated with considerable disturbance of speech, typified by difficulty in finding the right word to say, and sometimes by his saying the wrong word when attempting to say another word, and by stammering. He had recently improved in this respect. In December, 1938, he had a fit, and had had several since, though none in the past three months. Examination revealed evidence of the site of the

depressed fracture on the right side of his head, left-sided hemiparesis, and a lower brachial plexus palsy on the same side.

Huntington's Chorea.

Dr. Hall finally showed a male patient, an invalid pensioner, aged forty-seven years. He had attended hospital in May, 1939, complaining of weakness and difficulty in walking, slowly increasing during the past six years, and, during a slightly shorter period, of the development of writhing movements in the head and arms and difficulty with speech. His early history was obtained from the patient or the patient's wife. He was born in England and lived as a boy in London. He ran away at an early age, and joined the Navy. He came to Australia on the H.M.S. *Powerful*, and since then he had lost all contact with his family in England, and knew nothing whatever of any importance in regard to his family history. He left the Navy at the age of twenty-two years and became a trombone player in an orchestra. He followed the profession of music with a considerable degree of success until six years prior to the meeting, when his present symptoms commenced, and he soon had to abandon his music. Prior to the onset of his symptoms he was a model husband and father. He drank excessively at times, but was not troublesome. During the past six years he had undergone some mental degeneration. He was abnormally jealous, and at times very irritable and occasionally violent, though so far his violence had confined itself to acts of simple destruction, and had not been directed towards individuals. He was unable to concentrate and his memory was degenerating. He had a wife and two children alive and well.

The patient exhibited grimacing movements of the face and choreiform movements of the neck and arms. The facial movements were fairly constant, but the arm movements appeared only under stress or excitement; occasionally, in similar circumstances, slight movements of a similar type were seen in the legs. His gait was noteworthy: he walked on a wide base with his legs very slightly flexed at the knees, and his body slightly flexed at the hips. His shoulders were held more or less rigid; the arms were held to the side of his chest, whilst the right forearm was supinated and slightly abducted, and the hand, which was slightly flexed at the wrist, was held forward with the thumb and index finger opposed and the remaining fingers slightly flexed. On the left side the forearm was moderately flexed at the elbow, the hand was extended at the wrist and the fingers were flexed. In such an attitude he progressed with a curious spastic waddle. His speech, which suggested a spastic dysarthria, was in addition somewhat "explosive". His remarks, which were likely to be made in such manner, were punctuated by grunts and grimaces.

Examination further revealed a moderate generalized spasticity. The tendon reflexes were all very active and the plantar reflex on both sides was flexor in type. The abdominal reflexes were present and there were no sensory disturbances. Examination of the optic fundi and further neurological examination revealed no abnormality. The choreiform movements, which especially involved the patient's head, neck and arms and to some extent his legs, together with the mental degeneration, seemed to place him in the category of Huntington's chorea. In addition he had pyramidal tract degeneration on both sides, so that evidently he was undergoing extensive cerebral changes. Dr. Hall remarked that it was regrettable that he knew nothing regarding his family history.

Procidentia Uteri.

DR. M. BRITNELL FRASER showed a nulliparous patient, aged eighty years, suffering from extreme *procidentia uteri*. She had been seen by him one year earlier, complaining of frequency of micturition and of the sense of "a lump in her passage"; examination revealed then only very slight sagging of the anterior vaginal wall. One month prior to the meeting she complained of "complete falling of her womb" for one month, after a slight fall. Examination then revealed complete procidentia, with complete extroversion of both anterior and posterior vaginal walls. The introitus was small and the extruded mass had extreme venous congestion. Dr. Fraser explained that this condition was rare, and was due to some congenital defect in the musculature and connective tissue of the pelvic floor, which did not become apparent until late in life. Operative treatment was both difficult and unsatisfactory, because of the narrow entrance to the vagina, and he proposed to fit the patient with a small cup and stem pessary, which might give her relief.

Bilateral Ovarian Cysts with Amenorrhœa.

Dr. Fraser's second patient was a woman, aged thirty-six years, who complained of amenorrhœa of two months' duration and dyspepsia of four months' duration. She had had irregular menstrual periods for years, with intervals of amenorrhœa of two to four months. Examination revealed an irregular cystic mass filling the lower part of the abdomen and completely filling the pelvis. The cervix was extremely preaxial and above the level of the lower border of the *symphysis pubis*, and a small uterus could be felt bimanually above the brim of the pelvis and in front of the irrregular mass. An X ray examination of the pelvis revealed no fœtal parts. Dr. Fraser thought that the patient might have a dermoid cyst of one ovary and an innocent neoplasm of the other ovary, but he mentioned that Krukenburg's tumour could not be excluded. There was, however, no sign of ascites, and the patient's general condition was good.

Post-Graduate Work.

WEEK-END COURSE IN SURGERY AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine will hold a course of instruction in surgery at the Prince Henry Hospital, Little Bay, during the week-end May 11 and 12, 1940. The programme is as follows:

Saturday, May 11.

9.30 a.m.—In Ward 22, cases of interest: The Director of Post-Graduate Surgery, Mr. H. R. G. Poate.
10.30 a.m.—"Intravenous Anæsthesia", Dr. H. J. Daly.
11.15 a.m.—"Cystitis and its Treatment", Dr. R. J. Silverton.
12 noon.—"Acute Cholecystitis", Dr. V. M. Coppleson.
2 p.m.—"Acute Osteomyelitis", Sir Robert Wade.
3 p.m.—"Causes, Diagnosis and Treatment of Leucorrhœa", Dr. R. Davies.
4.15 p.m.—"Maxillary Sinusitis", Dr. Huff Johnston.
5 p.m.—"Infections of the Hand" (with film), Dr. J. C. Storey.

Sunday, May 12.

9.30 a.m.—"The Surgical Treatment of Hæmorrhage associated with Gastro-Duodenal Ulceration", illustrated by lantern slides, pathological specimens *et cetera*, Dr. H. C. R. Darling.
10.30 a.m.—"The Commoner Conditions Suitable to Radium Therapy", Dr. L. S. Loewenthal.
11.15 a.m.—"Injuries to Joints", Dr. Wilfred Vickers.
12 noon.—"Some Commoner Surgical Conditions of the Head and Neck" (with slides), Dr. A. J. Aspinall.

The lectures will be held in the lecture hall. A second week-end course in surgery will be held at the Prince Henry Hospital during the week-end August 3 and 4, 1940, a programme in regard to which will be published later.

The fee for either of these courses will be one guinea. Applications for registration, which must be accompanied by a remittance for the amount of the fee, must be made to the Secretary, New South Wales Post-Graduate Committee in Medicine, the Prince Henry Hospital, Little Bay.

Correspondence.

HARELIP AND CLEFT PALATE.

Sir: In the issue of this journal for March 16, Mr. K. B. Fraser published an article on harelip and cleft palate. This thesis I read with great interest and with great appreciation of the manner in which Mr. Fraser had blended embryological, anatomical and clinical details into a coherent story after the pattern of the best scientific presentations. I am not competent to comment upon the clinical side of the thesis, but I would like the opportunity of offering a few observations upon the embryological and anatomical aspects.

Mr. Fraser embraces what he calls the "classical embryological conception"—that the median nasal process is responsible for the formation of the median part of the upper lip, the philtrum and the median part of the upper alveolar process; and he states that he is unable to accept Professor Frazer's "radical departure" from this conception. In truth, the view which is attributed to Professor Frazer was first adumbrated by G. W. Callender (*Philosophical Transactions of the Royal Society*, Volume CLIX) in 1869, and it has been supported by the subsequent observations of Wood Jones ("Man's Place among the Mammals", 1929), Ashley-Montague (*Quarterly Review of Biology*, Volume X, 1935), E. H. Johnson (*The Journal of Anatomy*, Volume LXXI, 1937), and others, and in my opinion it is not so lightly to be cast aside.

Briefly, the view of these authors is that the maxillary processes of the embryo meet in the mid-line in front of the median nasal process, which becomes completely submerged; thus the median portion of the upper lip, the philtrum and the median portion of the upper alveolar process are all derived from the maxillary processes, not from the median nasal process. And, as is well known, there is no maxillo-premaxillary suture on the face, although there is such a suture on the hard palate, of course. This submergence of the premaxillæ by the maxillæ is a purely human characteristic, but incipient stages of submergence may be detected in the anthropoid apes, and in some human races the process is not wholly complete, as Wood Jones and Johnson have shown. Thus in some aboriginal Australians, for example, the inferior narial margin is rounded as in apes, or it is doubled, the posterior crest representing the narial margin of the premaxillæ, the anterior crest that of the overlying maxillæ. Moreover, in some embryos, and occasionally in the adult, the line of maxillary overlap is indicated by a suture which crosses the upper alveolar border obliquely towards the mid-line. Wood Jones and Johnson attribute the subnasal prognathism of the more primitive human races to incomplete submergence of the premaxillæ by the maxillæ, and it will be recalled that an exaggerated subnasal prognathism is one of the most striking features of bilateral harelip and cleft palate.

I am at a loss to understand why Mr. Fraser refuses to entertain this view. If the maxillæ have merely to join the sides of the premaxillæ, as he assumes, why do they not do so in any case? Or why should the premaxillæ protrude to such an extent? On the other hand, if the premaxillæ are normally submerged by the maxillæ, it is readily conceivable that too rapid growth of the premaxillæ carries these forwards before the maxillæ have an opportunity to meet and retain them within normal bounds. In other words, on the "classical" conception, however far the premaxillæ protrude their position between the maxillæ remains the same, and there is no reason why the three elements should not fuse; on the other hand, on the view presented here, such protruding premaxillæ prevent a normal union of the maxillæ, and these do not unite with an abnormal partner, the premaxilla; hence the cleft. This appears to be perfectly consistent with the condition usually found in harelip and cleft palate cases, and certainly it is worthy of consideration equal to that accorded to the "classical" view.

One other point: Professor Wood Jones has recently shown (*Journal of Anatomy*, Volume LXXIV, 1940) that the pharynx does not extend above the level of the palate, that the so-called oro-pharynx belongs to the nasal cavities; thus what is usually called the pharyngeal isthmus should be termed the *hiatus naso-pharyngeus*, as Mr. Fraser suggests. Wood Jones has shown further that the muscular band which forms the sphincter of the hiatus ("palato-pharyngeal" or "pharyngo-palatine" sphincter) and produces Passavant's "bar" or "ridge", is really a separated portion of the palato-pharyngeus muscle that has become incorporated in the superior constrictor of the pharynx; what in human anatomy is termed palato-pharyngeus is really the palato-thyroideus.

Yours, etc.,

A. A. Abbie.

Department of Anatomy,
University of Sydney,
March 20, 1940.

MEDICAL PRACTICE IN NEW SOUTH WALES: A DEGRADATION.

Sir: Under date February 29, 1940, the Executive Council of the State of New South Wales has approved of and promulgated most astounding regulations allowing unregistered persons to advertise that they are competent to treat human ailments.

They can advertise up to twelve inches of single-column space in any one issue of a newspaper, presumably as often as they like; they can advertise commencement or removal or sale or purchase of a "practice" up to twelve inches of single-column space, and are permitted to announce the removal of practice, the sale or purchase of a practice by means of a letter sent to their "bona fide patients". The words bona fide are rather intriguing when speaking of patients of unregistered persons. An unregistered person is also permitted to broadcast his capabilities.

He may also occupy up to four square feet of space in affixing two metal plates or notices, setting out his occupation, on the outside of his premises; but the regulation governing this naïvely prohibits a plate or notice exceeding two feet in length.

He is allowed to treat any human ailment or physical defect, but is not allowed in his advertisements or metal plates or notices to refer to cancer, tuberculosis, epilepsy, diabetes or venereal diseases. By inference, presumably, he may advertise that he is prepared to treat tumours, wasting diseases, fits and "private diseases".

It may be noted in this State that only qualified legal practitioners or conveyancers are permitted to do legal work for fees. It is an offence for any person to receive fees for treatment of sick animals, other than a registered veterinary surgeon. One cannot employ an electrician or plumber to do any work unless he has passed through a regular course of instruction in his calling and has been approved and registered. A builder is not allowed to use scaffolding on a building unless it has been passed by a competent inspector. We all know the intense amount of study necessary and examinations for fitness for those qualifying to become marine officers or engineers, and one can multiply these few instances. But when it comes to the human sick and disabled, any Tom, Dick or Harry can set up that he is competent to treat them and charge fees and be protected by these regulations.

One need not stress in a medical journal the countless number of sick whose recovery has been long delayed or rendered impossible by being treated by these ignorant people—every medical practitioner has seen examples. But now the law of the land really appears to give them a status.

The travelling unqualified practitioner has been an absolute menace in country districts, spending a few days here and advertising in advance his approaching visit to the town and being available to treat anything and everything, then moving on to the next town, and so on. A group of five daily newspapers in the north of New

South Wales realized this menace and markedly restricted their activities—for many years these newspapers have refused to accept advertisements from unqualified medical practitioners.

But now the legislature of New South Wales has put the imprimatur of its approval on unregistered medical practitioners.

Yours, etc.,
T. W. Lipscomb.

135, Macquarie Street,
Sydney,
March 15, 1940.

GHON'S PRIMARY FOCUS.

Sir: Dr. Colin Macdonald's discussion in your issue of March 2, 1940, of tuberculous infection of infants and children is of great interest. Will you please allow a few additions?

Anton Ghon, of Prague, began his investigation in 1903; reference was made to his findings in the *Wiener Klinische Wochenschrift* in 1907 and again in 1909; he published his book *"Der Primäre Lungenherd bei der Tuberkulose der Kinder"* in Berlin and in Vienna in 1912. This was quoted in an article in *The Australasian Medical Gazette*, on tuberculosis of the bronchial lymph glands, on May 16, 1914. The book was translated into English in 1916, and a review, based on a comparison of the English and German editions, was published in The Medical Journal of Australia (Volume I, 1916, page 398).

Ghon based his work on that of Parrot, who had announced his law that in the lung, as elsewhere, infection travels from the parenchyma to the corresponding lymph glands, and that infection of a lymph gland always implies an infective focus in the periphery and in that part drained by the gland. Parrot had communicated this to *La Société de la Biologie de Paris* in 1876; Ghon calls it *"la loi des adënopathie similaires de Parrot"*.

Ghon honours as his second inspirer G. Küss, whose *"De l'Hérédité Parasitaire de la Tuberculose Humaine"* appeared in Paris in 1898; and as his third, Birch-Hirschfeld, of Berlin, 1899. He does not appear to quote Cohnheim. His book is beautifully printed and illustrated by very clear diagrams and two magnificent coloured plates.

Ghon concludes that the presence of a primary focus in the lung, as he had demonstrated in tuberculosis of the tracheo-bronchial lymph glands, and the non-occurrence of retrograde infection from gland to lung proves that tuberculous pulmonary disease in infants is habitually aerogenous.

Yours, etc.,
Guy Griffiths.

131, Macquarie Street,
Sydney,
March 26, 1940.

Obituary.

WILLIAM JOHN RUSSELL.

We are indebted to Dr. Oliver Latham for the following appreciation of the late Dr. William John Russell.

The great Osler used to commence every chapter he wrote on disease with a short résumé of what older generations thought about it. Present-day medical men, whose work is marked by motor cars, aeroplanes, telephones, hospitalization, laboratories and other technical and electrical contrivances for diagnosis and treatment, may well pause to consider the life their grandfathers practised. William John Russell retired from active practice in West Maitland in 1905, when all these "modern contrivances" were just commencing. He had taken over in 1885 from his two brothers, who had associated with their relative, Dr. Blackwell, a little before.

Russell traced his ancestry to one Russell, an English adventurer, who in 1745, disgusted with the present state of England, had retired to remote County Clare. There he speedily espoused the daughter and co-heiress of Sheedy (Isoala) Macnamara, from whom he claimed connexion with the O'Briens and the royal house of Thomond. One son of this union was a rear-admiral in 1801. On his mother's side he was of the Norman and Catholic Skerretts (in Basque, Huscared), pleasantly described by Valentine Williams in his recent book "The World of Action". Sir William Stokes informs us that "even with the unlimited use of stratagems" making one's fortune was not an easy matter in Ireland in the nineteenth century. The father, escheated out of his father's farm by a younger brother, took note of the fact that Dublin houses with a bad or no title "went cheap". His native legal acumen enabled him to size up correctly the real powers of ownership professed by the sellers, and he bought these in wholesale manner and to such purpose that he became wealthy and actually resold some to the civic authorities when schemes of beautification developed, and with titles drawn up by himself. This parent married four times! However, a day came when a defaulting architect offered in part payment of his debts to apprentice the young William; and so in due course we find the latter designing and building various breweries and chimney stacks in England's "black country", which the proud architect in after years loved to assure us both "brew and drew" fine. However, his heart was in medicine, and soon he is to be found a resident student of Steven's (Swift's) Hospital, Dublin, with Sir Thomas Myles as superintendent, and with such men as the knight McDonald, the younger Colles, Swanzey, Purser, McAlister, Heuston and Corrigan as teachers. He was in residence there when the victims of the Phœnix Park murders were admitted. The sense of touch was called upon for much then. Colles had diagnosed a lump under the clavicle as an aneurysm, while a surgeon of the rising generation plumped for inflammation. The lance at first revealed laudable pus, but the "and now Mr. Colles" was, alas, speedily followed by Colles's "and now Mr. Smith" as bright red blood gushed forth and displaced the yellow streak. Mr. Colles, dressed in the usual frock coat, would with the utmost solemnity poke his forefinger into the carbolic spray. We can imagine a scene before X ray days when a railway shunter has had a heavy blow below the knee. No movement can be detected by the group of consulting surgeons. However, the sharp architect has noted the appearance of a transverse lineal groove during their manipulations, and they all accept his diagnosis, "impacted fracture of the upper end of the tibia". A day comes when the beloved teacher assumes a white and washable coat. The sympathetic students look aghast, since their native shrewdness, knowing their man, foresee impending dissolution. He died shortly after.

Russell's entry into Sydney's harbour was dramatic. A violent storm thereabouts had completely dismasted their ship, and when battened down the captain rushed in and told them all they had but ten minutes to live. Immigrants wail, children scream and men, according to their way, carry on. The doctor can be imagined calling out his crest's motto, *"che sara sara"*—what will be, will be! However, the winds moderated and a friendly tug appeared. Curiously enough, another emigrant ship was found to be in the same predicament and quite close to them.

His first case in Maitland was a fractured spine. On a great black racehorse almost daily for six weeks he rode to Mount Vincent, nearly twenty miles away, till danger was past and the patient convalescent. As early as 1888 he developed for himself the technique of attacking hydatids of the liver through rib resection. He had also the unenviable experience of having a patient die in his surgery after the old method of diagnosing hydatids by needling. He often harried the writer by demanding a scientific cause for the death phenomena here. Prodding a body cavity with a long needle of, say, five-millimetre

bore was common practice in those days. He gave clear evidence in court and was much sought after in will cases. A man had been shot in the face by quail shot and the judge had most improperly taken the side of the defending barrister. "Are you quite certain, Dr. Russell, that such wounds might not have been caused by a charge of salt?" The doctor merely answered: "I can only declare, Your Honour, he was well peppered!"

He had a most uncanny capacity for seeing the "devil" in a person's nature or danger signals in disease. He was the centre of an unpleasant episode wherein what he diagnosed as physiological his *confrères* adjudged pathological. The abdominal operation brought a welcome addition to Australia's census, but his caustic remarks only brought on himself a round robin denying him any cooperation henceforth. However, a marvellous matron ruled the hospital, and with her aid all previous records for major hospital operations went by the board. The operations commonly performed in simple "inns" in those days before private hospitals abounded would surprise the present generation, and the total cost, board and all, might easily be below that of the mere anæsthetic today. During his year of "Coventry" he placed a man with strangulated hernia on a hotel sofa and, placing two candlesticks on his abdomen, assured him that if he moved the resulting darkness might be his death. He then proceeded to relieve the strangulation, and, sweeping away redundant tissue, a few well-placed stitches achieved a radical result. The spirit of rivalry was rampant in those days. A Sydney knight who commenced life as a surgeon was appalled at a conglomeration of wens on a woman's head and just played with one of them. She came to Maitland and at one sitting had all thirteen removed. Nearly everything was done in the surgery in those days. A pleasing aftermath was a letter of praise from the gallant knight after he had received his patient back.

Since his fortune went well past six figures, a glance at his business faculties might not be amiss. He often quoted the late Mr. L. Bruck's advice to doctors: (i) Never get in on the ground floor on any account in any new company formation. (ii) Never buy shares in a company without large assets disclosed. (iii) Never invest where the company had not paid dividends the last three years. This was Mr. L. Bruck's experience of doctors, as he sized up their practices for sale and listened to many a weary tale of woe. Six shrewd business men tried to get Russell to join them to buy from a chronic alcoholic his tontine policy of several thousand pounds. Russell's answer was: "He eats well, he exercises and is well cared for; he'll see you all out." Also he consulted the local "A.M.P." agent, who warned "Look up the policy", which only promised bonuses to the insured himself. The deal went through, and the next five yearly bonuses provided only a "fiver" in lieu of the previous hundreds from this American company; and the man did outlive most of them. A grateful, if poor, patient could only show his gratitude by presenting a 17-foot snake, together with some rabbits, to show the snake's proficiency and agility. The local sergeant of police, also agent for the Society for the Prevention of Cruelty to Animals, paid a surprise visit and expressed genuine interest in the snake's sleight of jaw and coil. The worthy medico, however, smelt a rat, and putting his ears back, assumed a most natural deafness. The old Dublin school of medicine knew the value of Epsom salt for eye lotions, the depressant qualities of castor oil, tincture of iodine and of iron for cutaneous applications, and one wonders if their application of caustic antimony for eastern venereal sores may not have been based on some clinical observation, because, for cheapness of board, many stricken soldiers from India were lodged in Dublin. It will be remembered too that Steven's Hospital had always one of the few lock wards existing in the British Isles. Relieving dispensing chemists maintained that his doses were on average 50% larger than the usual custom. He was not afraid of his judgement of a patient's capacity to withstand powerful drugs, especially in the mental and neurotic sphere. Curiously enough he always gave a shocked injured man a drachm of *Liquor Morphinæ*, a dose (three-fifths of a grain) recommended in the Navy in the Great War. On occasions he would repeat this almost indefinitely. He recalls how his predecessor treated incurable headache by shaving the head, applying a mustard poultice, washing, then warm milk and, lastly, three dozen leeches. "The pain went."

His first wife was a daughter of Mr. Samuel Clift, of East Maitland; but he lost both wife and child. He afterwards married Marian, daughter of Joseph Pearse, of "Tivoli" (now Kambala C.E. girls' school) and various New England stations, by whom he had Major Rivers Russell, M.C., R.A.; Charles, a captain in the Duke of Cornwall's regiment; William, late H.M.S. *Australia*; two other sons, and Mrs. Job, who has two sons at Knox Grammar School. A nephew, Major-General W. R. Blackwell, C.M.G., R.A.M.C., was second in command at Whitehall, while another nephew, Colonel Frizell, of the South Staffords, was Deputy Colonel, acting General at thirty-one, and gained the M.C. and bar and D.S.O. His brother, Colonel George Blakeney Russell, R.A.M.C., who preceded him in Maitland, gained the travelling scholarship and was bracketed first in the medical finals at Trinity College, Dublin, about 1881, with the late Father Pigot, the famous seismologist at Riverview College, Sydney. Another nephew was the fastest "runner" on the western front, at least in the British Army. A good story is told of this runner's maternal grandfather, Major Dowie. At ninety-two he used to take his daily walk of a mile or so with Russell in Ireland. In 1914 all regiments employed people to cable all over the world for ex-officers to return and join up. Of course, one was sent to this nonagenarian and the gallant Irishman answered with spirit: "I'll come if you send a cab for me!"

Before deciding to invest more money in any company, Russell would walk round and round the main offices *et cetera* of such institutions as Tooth's, the "B.H.P.", Australian Glass Company, Colonial Sugar, John Brown's, East Greta Coal, and some new bank branch opened, and if the volume of trade seemed propitious, the goods up to standard and reports from far and wide satisfactory, he would both venture more himself and advise his friends likewise. It is difficult to adequately give any idea what he meant to the Maitland district forty years ago. On a recent trip to Barrington Tops old patients of his came to meet us right to the last inhabited house on the upper Allen River.

BARON BROOKE.

We regret to announce the death of Dr. Baron Brooke, which occurred on March 31, 1940, at Sydney, New South Wales.

Proceedings of the Australian Medical Boards.

SOUTH AUSTRALIA.

The undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1919 *to* 1935, of South Australia, as duly qualified medical practitioners:

Mullany, John Charles Colahan, M.B., B.S., 1938 (Univ. Melbourne), Rose Park.

Laing, John Joseph, M.B., B.Ch., 1924 (Univ. Dublin), University of Adelaide, Adelaide.

Donaldson, William Edward, M.B., B.S., 1925 (Univ. Melbourne), Adelaide.

The following additional qualifications have been registered:

McCoy, Harold Alexander, F.F.R. (London), 1939, Thorngate.

Bonnin, Josiah Mark, M.R.C.P. (London), 1939, Adelaide.

Wurm, Bertram Eric, D.G.O. (Trinity), 1935, L.M. (Rotunda), 1935, D.C.O.G. (British College), 1935, Prospect.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Act of* 1939, of Queensland, as duly qualified medical practitioners:

Gill, Robert Chalmers, M.B., B.S., 1939 (Univ. Sydney), District Hospital, Mackay.

Leggett, Clarence Arthur Campbell, M.B., B.S., 1937 (Univ. Sydney), District Hospital, Cairns.

McGregor, Alexander Roy, M.B., B.S., 1938 (Univ. Sydney), District Hospital, Ipswich.

The following additional qualification has been registered:

Watkins, Eric Roderick, Ch.M., 1939 (Univ. Sydney), District Hospital, Mount Morgan.

Notice.

CLINICAL DEMONSTRATIONS IN SYDNEY.

A MEETING of the Medical Board of the Royal Prince Alfred Hospital will be held on Thursday, April 11, 1940, in the A2 Lecture Theatre. The clinical section will last approximately one hour and will commence at 4.30 p.m. sharp. Members of the British Medical Association are invited to be present.

A detailed programme of the cases to be presented is as follows:

1. Demonstration of a case of neurogenic sarcoma of the skin, with specimens of two cases, by Professor H. R. Dew.

2. Clinico-pathological demonstration of a specimen showing ureteric implantation into the colon for incontinence of urine in *spina bifida occulta*, by Professor H. R. Dew and Dr. G. Davies.

3. Demonstration of a case of injury to the common bile duct with repair two years ago, by Mr. J. S. MacMahon.

4. Demonstration of a case of unilateral lymphœdema of a lower extremity, spontaneous subarachnoid hæmorrhage and plexiform angioma of the *cauda equina*, by Mr. Gilbert Phillips.

A CRICKET MATCH BETWEEN DOCTORS AND DENTISTS.

THE annual cricket match between teams representing the dental and medical professions in Sydney was played at the Sydney Cricket Ground on February 14, 1940. According to the generally accepted laws of cricket the match resulted in a draw; but according to a rule agreed to by the rival captains (R. H. Bettington and C. S. Winning) before the commencement of play, the medical men had a hollow victory. The agreement was that the team scoring the most runs in the time allotted to it was to be the winner, regardless of the number of wickets that fell.

The medical men batted first, and largely as the result of attractive and forceful batting by Truscott, Delohery, Bettington and Halliday, made 272 runs for the loss of five wickets in two hours. In the two hours remaining for play the dentists made 152, losing 9 wickets. Taylor (49) and Stenmark (38, not out) batted attractively. In the closing stages chief interest centred on the medical men's efforts to dismiss N. I. Blue. The efforts were unsuccessful; in fact, they appeared at no time to have any promise of success.

The proceedings throughout the afternoon, especially the final hour, were enlivened by the comments, favourable and otherwise, of men of the Second Garrison Battalion, who are quartered at the Sydney Cricket Ground. They applauded vigorously when applause was merited; they howled with delight when a catch was missed; they advised fieldsmen to get bags; and when N. I. Blue was batting and the field was set close to the wicket, they instructed the fieldsmen to "give him air".

An endeavour is being made to secure a suitable shield to be played for annually and to be retained for the succeeding twelve months by the winning team. It has been suggested that it might be called the Barbour-Hardwicke Shield, to perpetuate the names of two cricketers—a medical man and a dentist respectively. During this year's match a sum of £5 9s. was collected from the participants. Any medical man who wishes to contribute should communicate with Dr. J. K. Brooks, 201, Elizabeth Street, Sydney.

Scores.

Doctors: First Innings.

M. S. Truscott, stpd., b. Stenmark	78
J. R. O. Roger, b. Winning	8
J. Delohery, c. Taylor, b. Stenmark	56
R. H. Bettington, c., b. Cusick	61
G. C. Halliday, not out	44
R. Mills, b. Turnbull	7
K. Priddis, not out	9
Sundries	9
Five wickets for	272

(Innings closed.)

Bowling: C. S. Winning, 1 for 50; S. M. Turnbull, 1 for 67; A. Lawes, 0 for 28; J. O. Stenmark, 2 for 51; N. I. Blue, 0 for 38; M. Cusick, 1 for 29.

Dentists: First Innings.

A. Lawes, b. Calov	5
W. Jones, l.b.w., b. Bettington	10
J. M. Taylor, b. Lawes	49
M. Cusick, c. Calov, b. Lawes	0
G. Saggers, l.b.w., b. Lawes	11
R. Curtin, c. Roger, b. Bettington	9
O. Keyte, c. Roger, b. Bettington	3
J. O. Stenmark, not out	38
S. M. Turnbull, c. Priddis, b. Lawes	15
C. S. Winning, c. Lawes, b. Bettington	6
N. I. Blue, not out	4
Sundries	2
Nine wickets for	152

Bowling: W. L. Calov, 1 for 25; C. H. Lawes, 4 for 45; R. H. Bettington, 4 for 44; J. R. O. Roger, 0 for 24; B. W. Stevenson, 0 for 12.

Books Received.

DEMONSTRATIONS OF PHYSICAL SIGNS IN CLINICAL SURGERY, by H. Bailey, F.R.C.S.; Seventh Edition, revised; 1940. Bristol: John Wright and Sons Limited. Medium 8vo, pp. 322, with 377 illustrations, some of which are in colour. Price: 21s. net.

A TEXT-BOOK OF X RAY DIAGNOSIS, by British authors, edited by S. C. Shanks, M.D., M.R.C.P., F.F.R., P. Kerley, M.D., M.R.C.P., F.F.R., D.M.R.E., and E. W. Twining, M.R.C.S., M.R.C.P., F.F.R., D.M.R.E.; Volume III; 1939. London: H. K. Lewis and Company Limited. Crown 4to, pp. 814, with 710 illustrations. Price: 63s. net.

THE SYNOVIAL MEMBRANE AND THE SYNOVIAL FLUID, WITH SPECIAL REFERENCE TO ARTHRITIS AND INJURIES OF THE JOINTS, by D. H. Kling, M.D.; 1939. London: Baillière, Tindall and Cox. Medium 8vo, pp. 284, with 80 illustrations. Price: 25s. net.

A PRIMER OF DIETETICS, by W. A. Osborne; Fifth Edition; 1940. Melbourne: W. Ramsay (Surgical) Proprietary Limited. Demy 8vo, pp. 55. Price: 2s. 6d. net.

MANUAL OF FRACTURES, DISLOCATIONS AND EPIPHYSEAL SEPARATIONS, by H. C. W. S. de Brun, M.D., F.A.C.S.; 1940. Chicago: The Year Book Publishers Incorporated; Melbourne: W. Ramsay (Surgical) Proprietary Limited. Demy 8vo, pp. 468, with illustrations. Price: $3.50 net.

MYTHOLOGY OF THE SOUL. A RESEARCH INTO THE UNCONSCIOUS FROM SCHIZOPHRENIC DREAMS AND DRAWINGS, by H. G. Baynes, M.B., B.C.; 1940. London: Baillière, Tindall and Cox. Medium 8vo, pp. 952, with 53 illustrations. Price: 32s. 6d. net.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Cumming, Gordon David, M.B., B.S., 1936 (Univ. Sydney), 42, Victoria Parade, Manly.

Howle, Don Creswell, M.B., 1938 (Univ. Sydney), 142, William Street, Bathurst.

Mills, Roy Markham, M.B., B.S., 1939 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Diary for the Month.

APR. 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee; Organization and Science Committee.
APR. 9.—Tasmanian Branch, B.M.A.: Branch.
APR. 12.—Queensland Branch, B.M.A.: Council.
APR. 16.—New South Wales Branch, B.M.A.: Ethics Committee.
APR. 17.—Western Australian Branch, B.M.A.: Branch.
APR. 18.—New South Wales Branch, B.M.A.: Clinical Meeting.
APR. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
APR. 24.—New South Wales Branch, B.M.A.: Branch.
APR. 24.—Victorian Branch, B.M.A.: Council.
APR. 26.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. Margaret B. Cleland has been appointed Temporary Honorary Assistant Physician at the "Mareeba" Babies' Hospital, South Australia.

* * *

Dr. H. M. Jay has been appointed an Honorary Consulting Aural Surgeon at the Royal Adelaide Hospital, Adelaide, South Australia.

* * *

Dr. R. McMahon Glynn and Dr. A. S. de Bohun Cocks have been appointed Honorary Aural Surgeon and Honorary Assistant Aural Surgeon at the Royal Adelaide Hospital, Adelaide, South Australia.

* * *

The following appointments have been made at the Royal Adelaide Hospital, Adelaide, South Australia: Temporary Honorary Physicians: Dr. F. H. Beare, Dr. F. R. Hone, Dr. K. S. Hetzel; Temporary Honorary Assistant Physician: Dr. R. T. Binns; Temporary Honorary Clinical Assistant to the Medical Section: Dr. Margaret B. Cleland; Honorary Anæsthetist: Dr. R. H. Elix; Temporary Honorary Assistant Ophthalmologist: Dr. T. L. McLarty.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii-xx.

ALFRED HOSPITAL, PRAHRAN, VICTORIA: Acting Honorary Surgeon.

ROYAL AUSTRALIAN AIR FORCE: Medical Officers.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Resident Medical Officer, Honorary Officers.

TARA DISTRICT HOSPITAL, TARA, QUEENSLAND: Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	Associated Medical Services Limited. All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Federated Mutual Medical Benefit Society. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	Wiluna Hospital. All Contract Practice Appointments in Western Australia.

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